DOCTOR OF PHILOSOPHY

Neuromuscular performance and exercise stress associated with the stabilisation of synovial joints

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NEUROMUSCULAR PERFORMANCE AND EXERCISE STRESS ASSOCIATED WITH THE STABILISATION OF SYNOVIAL JOINTS

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SUMMARY

The frequency of anterior cruciate ligament injuries (ACL) by non-contact aetiologies (Rees, 1994) and the potentially severe consequences for the team games player (Kujala et al., 1995) has underscored the need for understanding of the importance of neuromuscular mechanisms in the effective maintenance of knee joint integrity (Gleeson et al., 1998a). In particular, the knee flexor muscle group offers greatest dynamic protection against injuries to this ligament (Johansson, 1991). Traditional (volitional) methods of assessment of neuromuscular performance can be confounded by inhibitory mechanisms (Gleeson, 2001). Accordingly, the painless technique of magnetic stimulation of peripheral nerves has received increased attention for the assessment of aspects of 'true' neuromuscular capacity (King and Chippa, 1989). There is accumulating evidence that implies a linkage between fatigue and injury (Gleeson et al., 1998b; Hawkins et al., 2001). However, further investigation is required to explore how such exercise may affect 'true' performance capacity and the possible implications for knee joint stability. Estimates of indices of neuromuscular performance, such as electromechanical delay, rate of force development and time to half peak force, may offer important information concerning the temporal capabilities of the active joint stabilisers to initiate and muster meaningful levels of muscle force. These estimates may also provide an insight into the likely knee injury avoidance capabilities (Gleeson et al., 1998b; Mercer et al., 1998). However, only limited information is available in the contemporary scientific literature regarding the reproducibility and reliability characteristics of these indices of performance for the knee flexors, particularly subsequent to magnetic stimulation.

Study 1 (chapter 4)

This study investigated the reproducibility and single measurement reliability of indices of neuromuscular performance elicited during volitional and magnetically evoked activation of the knee flexors in males and females. Analyses showed equivocal measurement variability between sexes and that magnetically evoked indices of neuromuscular performance generally offered statistically equivocal levels of measurement reproducibility compared to traditional volitional methods ($p < 0.05$). Indices of volitional peak force (PF_V), electromechanical delay (EMD_V) and magnetically evoked electromechanical delay (EMD_E) offered the greatest practical utility for the assessment of neuromuscular performance requiring the fewest intra-individual assessment trials to achieve acceptable precision of measurement. However, apart from the index PF_V, single trial protocols assessing all other indices of performance offered poor measurement precision during intra-individual or intra-group performance comparisons. Furthermore, inter-day assessments were associated with a significantly inferior level of measurement reproducibility by contrast to intra-session assessments ($p < 0.001$).

Study 2 (chapter 5)

The second study examined the inter-day reproducibility and single measurement reliability of indices of voluntary and magnetically evoked neuromuscular performance of the knee flexors of males and females following an acute fatigue task. Analyses showed generally no difference in measurement reproducibility and single measurement reliability between modes of muscle activation (volitional; magnetically evoked), or between sexes. Furthermore, most indices of performance were not associated with an increase in variability of performance following acute

muscle fatigue compared to baseline. Some indices ($T_{½E}$, EMD_E) showed superior measurement reproducibility following fatigue tasks ($p < 0.01$). In a similar manner to the results for assessments involving un-fatigued performances, single trial protocols would have been unlikely to offer sufficient measurement precision during assessments of neuromuscular performance following fatigue, for either intra-subject or intra-group comparisons. The current data suggested that the indices PF_V and EMD_E offered the greatest practical utility for the assessment of post-fatigue neuromuscular performance requiring the fewest intra-individual assessment trials to achieve acceptable level of measurement precision.

Study 3 (chapter 6)

Female team-games players have been estimated to be at five to eight times greater risk of ACL injury by comparison to male counterparts (Gray et al., 1985; Hutchinson and Ireland, 1995). As such, the third study focussed on the effects of an acute intermittent bout of maximal intensity static exercise on the voluntary and magnetically evoked neuromuscular performance in the knee flexors of males and females to investigate any differential responses to fatigue. The fatigue intervention induced a substantive reduction in the volitional force generating capabilities of the knee flexors that was greater in males compared to females ($p < 0.01$), and an impairment in electromechanical delay performance (EMD_V) in females exclusively ($p < 0.05$). Such temporal impairment, coupled with decreased force generating capabilities, may be congruent with increased threat to knee joint stability. The ultimate physiological capacity of the neuromuscular system, as measured by magnetic stimulation, was either preserved ($T_{½E}$), or potentiated (EMD_E, P_TF_E, RFD_E) by a similar extent in both males and females ($p < 0.05$).

Preservation and potentiation of some indices of magnetically evoked neuromuscular performance may suggest that even under conditions involving fatigue, there remains the potential to access a greater proportion of the capacity of the neuromuscular system when a threat to joint stability is perceived.

Study 4 (chapter 7)

The effects on neuromuscular performance of an increase in the volume of fatiguing exercise was the focus of the fourth study. While study three had deployed a single episode of fatiguing exercise, the fourth study, involving four equivalent episodes, showed a cumulative decrease in the peak force (PF_V) capabilities of the knee flexors of collegiate female team-game players ($p < 0.05$). Investigation into the acute recovery showed almost total restoration of PF_V performance by six minutes. An associated increase in EMD_V following the first episode of exercise ($p < 0.05$) was maintained throughout the remainder of the intervention and recovery. Such fatigue-related EMD_V changes may place the female athlete at increased and prolonged risk of knee injury, since the temporal capabilities of the knee flexors to initiate muscle force may be fundamental to the prevention of anterior cruciate ligament (ACL) injury (Gleeson et al., 1998b). However, the potentiation of the (magnetically evoked) temporal capacity of the knee flexors following fatigue and throughout the recovery period may offer evidence of a vital neuromuscular compensatory strategy to overcome decrements to volitional performance capabilities. Accordingly, an episode of fatiguing exercise may actually facilitate performance and ultimately enhance the capability to resist joint injury.

Study 5 (chapter 8)

The fifth investigation examined the effects eccentric exercise-induced muscle damage (EIMD) and a static fatigue task in the knee flexors (agonist) on the voluntary and magnetically evoked neuromuscular performance of the agonist and antagonist muscle groups in males. The results strongly suggest that muscle damage was induced in the knee flexors (characterised by increases in soreness, plasma creatine kinase and decreases in peak force that were most prominent at 48 hours). Performance of the static exercise task during the EIMD condition induced further reductions to the contractile capabilities of muscle symptomatic of damage. However, since the greatest sole decrements to contractile capabilities were observed following eccentric exercise by comparison to the fatigue task, EIMD may be associated with greater relative impairments to the dynamic capability to resist knee injury. The effects of EIMD on the antagonist was limited to a minor decrease in RFD_V capabilities (at 48h). Such changes to the performance capabilities of the active knee stabilisers may present substantive challenges to the neuromuscular system's capability to protect the ACL during mechanical loading of the knee joint, especially at knee angles proximal to full extension. The evoked neuromuscular performance capacity of the knee flexors as derived from magnetic stimulation of the sciatic nerve, was maintained during conditions of EIMD and fatigue. Some indices of performance were even potentiated (EMD_E, $p < 0.01$) following the fatigue intervention. This preservation of neuromuscular performance capacity may help prevent injury on each occasion when the sports performer experiences fatigue and/or symptoms of exercise-induced muscle damage. However, the utility of this mechanism to the prevention of injury may be dependent entirely on routine neuromuscular inhibitory processes being

overwhelmed by those capable of offering a potentiation of performance at critical times of threat to joint stability.

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Chapter 1
Introduction

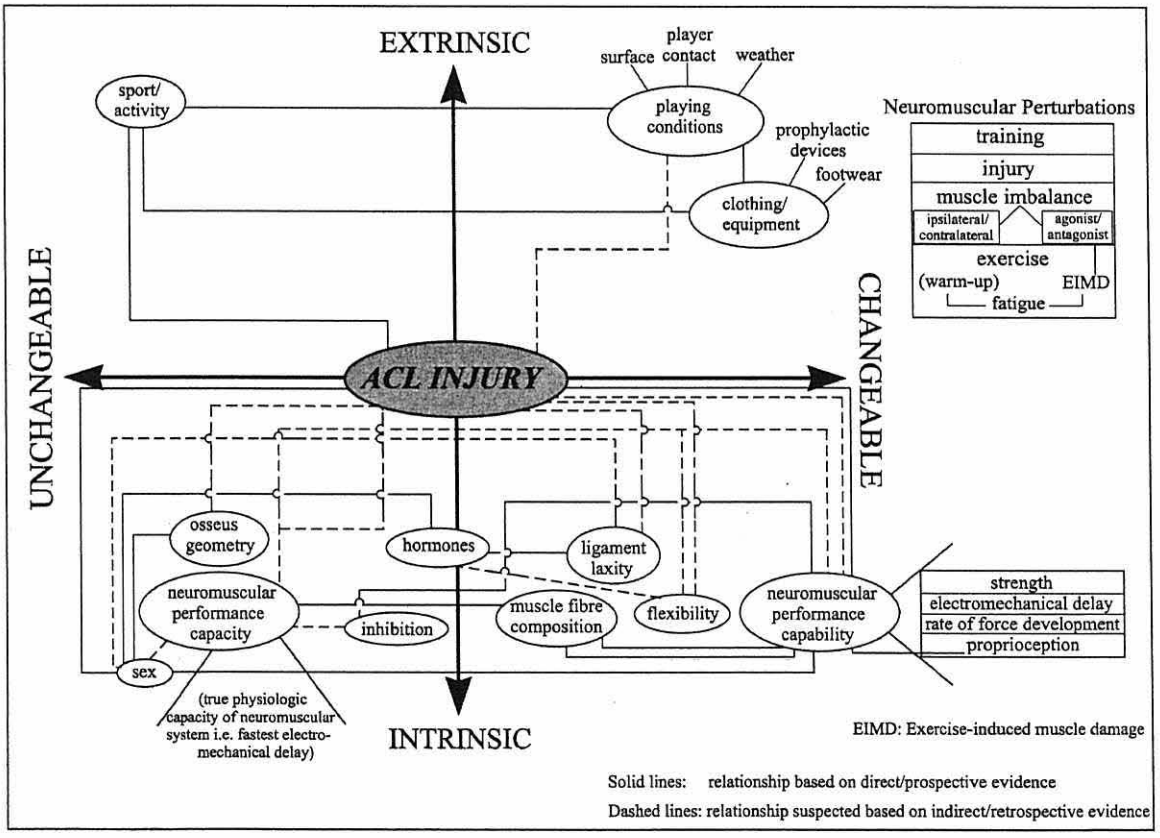
1.0 INTRODUCTION

1.1 *Knee joint stability and injury*

A conceptual model for knee joint stability depicts a complex interaction of passive structures (osseous geometry, ligaments, menisci and capsular structures) with the active (muscle) stabilisers (Johansson et al., 1991; Fu et al., 1993). The accumulating evidence of knee ligament injuries by non-contact aetiologies in team sports athletes (Rees, 1994; Hutchinson and Ireland, 1995; Ireland et al., 1997) underscores the importance of potential neuromuscular protective mechanisms of the active stabilisers. It has been estimated that injuries to the anterior cruciate ligament (ACL) represent half of all knee ligamentous injuries (Bollen, 1998) and has an incidence of 1 per 1000 participants in sport (Hirshman, 1990). Previous research has shown that the ACL is under greatest mechanical strain between 0 to 0.52 rad (29.8°) of knee flexion (0 rad = full extension) (Beynnon and Johnson, 1996). Thus, the knee joint positioned approximately in this range would be most vulnerable to adverse forces and injury compared to intermediate joint positions. Indeed, research shows that injuries to the ACL most often occur with the knee joint proximal to full extension (Gray et al., 1985; Rees, 1994; Ireland et al., 1997). The ACL is the principle ligamentous restraint to tibio-femoral rotation and anterior tibio-femoral displacement (Fu et al., 1993; Rees, 1994), accordingly, the most common mechanisms of injury often involve movement patterns incorporating rapid changes in direction and landing (Noyes et al., 1983; Hutchinson and Ireland, 1995; Ireland et al., 1997). The knee flexor groups are considered especially important in the active regulation of such joint movements and are the principle dynamic protector of this ligament (Fu et al., 1993; Rees, 1994; Gleeson and

Mercer, 1996). The implications of ACL injury to the sports performer can be severe. Untreated, sports related ACL injuries are the most common cause of permanent disability (Kujala et al., 1995). In addition, contemporary surgical interventions demand a subsequent rehabilitation programme of at least 6 months prior to the participant recommencing competitive match play (Rees, 1994; Shelbourne and Gray, 1997). While numerous factors have been implicated in the risk and incidence of ACL injury, recent research has highlighted the potential importance of neuromuscular mechanisms in the maintenance of knee joint stability. Figure 1.1 outlines these factors and how such are thought to be interrelated with other integral neuromuscular and musculoskeletal parameters.

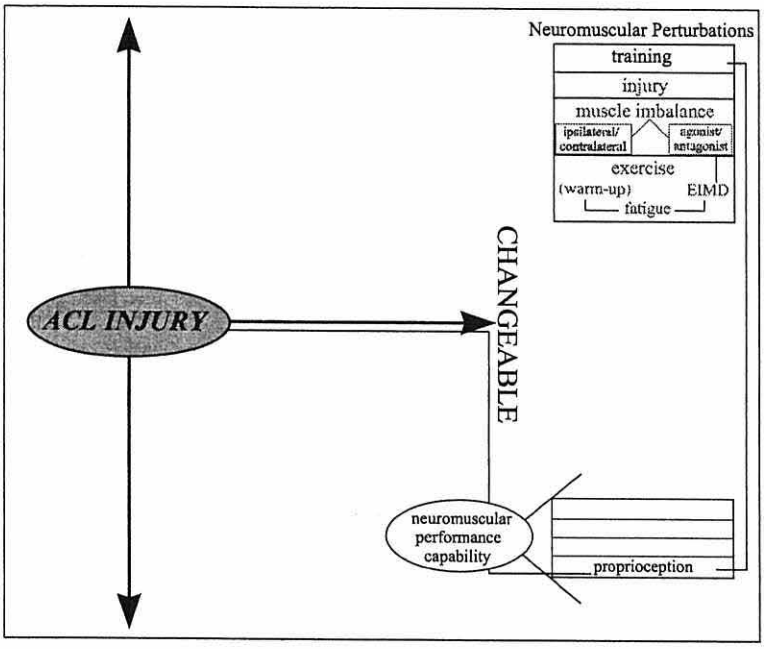
Figure 1.1. Locus of anterior cruciate ligament (ACL) injury.



1.2 *Injury prevention, neuromuscular performance and control*

Given the potentially severe implications of ACL injury, the development of propholactic interventions as a preventative strategy may be a primary goal of the sports medicine practitioner. Despite a substantial research focus on diagnosis and treatment of such injuries (Rees, 1994; Beynnon and Johnson, 1996; Roos and Karlsson, 1998), relatively little research has focussed on mechanisms that may contribute to the prevention of ACL injury. The few prospective randomised control trials that have investigated possible injury preventative strategies have predominantly focussed on proprioceptive training (Caraffa et al., 1996; Wedderkopp et al., 1999; Soderman et al., 2000). The most prolonged intervention (3 years) involving daily 20 minute wobble-board training sessions of increasing difficulty (n = 300), was successful in reducing the incidence of ACL injuries by seven fold compared to a control group (n = 300) in semi-professional soccer players. However, results from similar but shorter interventions (over one competitive season) report no significant differences (Wedderkopp et al., 1999; Soderman et al., 2000) in incidence of ACL injury in intervention groups by comparison to controls. From the limited evidence available, it appears that prolonged progressive proprioceptive training may have the potential to significantly impact dynamic joint protection and ACL injury.

Figure 1.2. Proprioception and ACL injury.

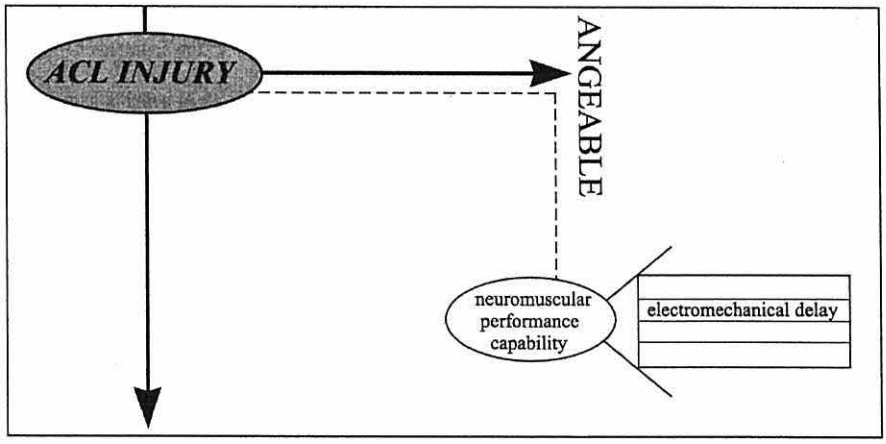


1.3 *Injury prevention, neuromuscular performance and speed of muscle activation*

A more recent definition of proprioception encapsulates the sensory aspects of performance, such as kinesthetic information regarding joint pressure and soft tissue movement, in addition to the motor system, which allows movement of the body to correct posture (Biedert, 2000). In order to protect effectively against ACL injury, imposed joint forces must be rapidly regulated by the neuromuscular system (Swanik et al., 1997). However, the neuromuscular system has a limited reaction time response to dynamic forces applied to the knee (Devita and Skelly, 1992, Gleeson et al., 1998a). Accordingly, any sensory motor corrections in response to kinesthetic perturbations will be subject to a time delay. The effectiveness of the protective capabilities of the active knee stabilisers may be related substantively to this time delay (Wojtys and Huston, 1996; Gleeson et al., 1998a; Mercer et al., 1998). One aspect of neuromuscular reaction time is electromechanical delay

(EMD), which represents the time delay between the onset of muscle electrical activity and the onset of tension development in skeletal muscle (Norman and Komi, 1979; Zhou et al., 1996). This index of performance alone is speculated to influence considerably knee joint stability. Such tenets are based on evidence from investigations showing significantly inferior EMD performance in ACL-deficient limbs by comparison to healthy controls (Gleeson et al., 1998b; Kaneko et al., 2002) and observations of subsequent improvements independent of other indices of neuromuscular performance following physical therapy interventions (Gleeson et al., 1998b). Furthermore, the substantive prolongation of this neuromuscular delay following muscle fatigue (Gleeson et al., 1997; 1998b; Mercer et al., 1998; Zhou et al., 1996) may ultimately permit the unrestrained development of forces of sufficient magnitude to damage knee ligamentous tissue (Mercer et al., 1998).

Figure 1.3. Electromechanical delay (EMD) and ACL injury.

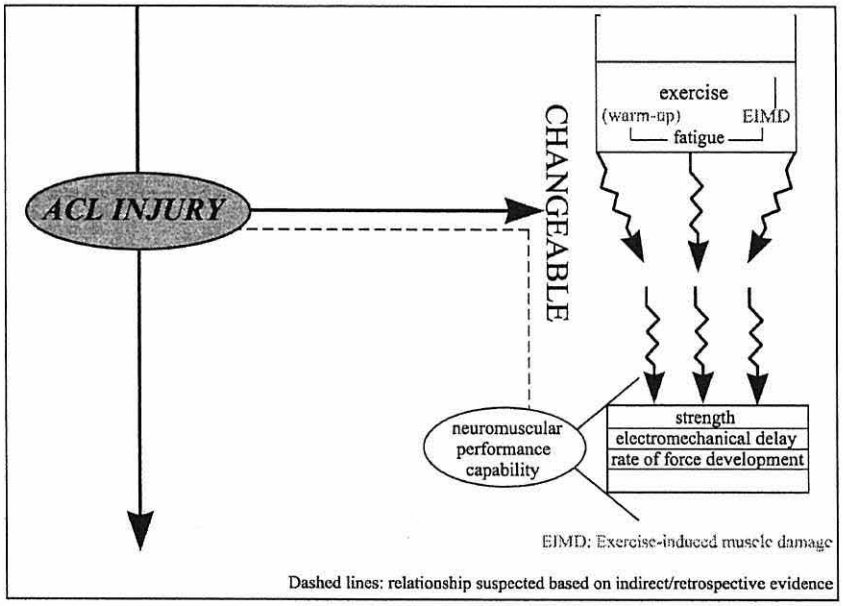


1.4 *Effects of exercise on electromechanical delay (EMD)*

Acute fatigue tasks, which may be representative of the physiologic demands of the short episodes of intense activity within team sports (McInnes et al., 1995) have been associated with a dramatic prolongation of EMD values of up to 42% to 45%

longer compared to pre-exercise levels (Horita and Ishiko, 1987; Zhou et al., 1996). Such temporal impairments are also typically accompanied by concomitant decrements to the capabilities to generate muscle force (e.g. 15% - 56% decrease in peak force (Yeung et al., 1999; Zhou et al., 1996)). However, the effects of acute single bouts of high intensity exercise may underestimate the potential neuromuscular impairments experienced by the team games player. For example, serial episodes of maximal acute exercise are frequently demanded of the sports performer during competitive match-play (Bangsbo, 1994; McInnes et al., 1995). Indeed, results from studies utilising protocols involving serial episodes of maximal exercise suggest the potential for greater decrements to EMD capabilities compared to a single bout (up to 70% longer compared to pre-exercise values) (Zhou et al, 1996). The 'net' effect of such temporal impairments in addition to concomitant subsequent reductions in the force generating capabilities of the knee flexors, may be the fatigue athlete has a substantively reduced 'margin for error' by which to elicit timely corrective neuromuscular strategies to kinesthetic joint perturbations, placing the athlete at increased risk of injury.

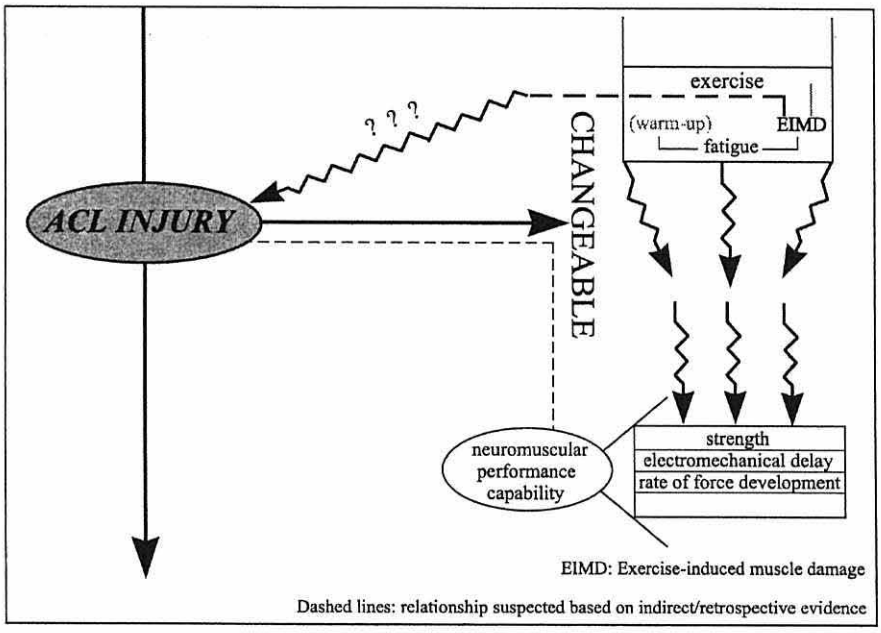
Figure 1.4. Possible interrelationships between muscle fatigue, neuromuscular performance capability and ACL injury.



Return to competitive match play following a prolonged period of inactivity or injury can render muscle susceptible to ultrastructural damage, particularly if the chosen activity involves a large eccentric component (Armstrong et al., 1983; Jones et al., 1989; Byrne and Eston, 1998). The subsequent exercise-induced muscle damage (EIMD) is typified by potentially large reductions in the force generating capabilities (e.g. up to 69% reductions in peak force (Rinard et al., 2000)) which may persist for several days following exercise (Rinard et al., 2000; Nokasa et al., 2001), in addition to a delayed pain response in the involved musculature (Foley et al., 1999; Rinard et al., 2000; Byrne et al., 2002). While the effects of EIMD on the susceptibility to further injury is relatively unknown, the findings of a recent Football Association audit of injuries of all professional soccer clubs which showed that 13% of all injuries sustained were during pre-season training (Hawkins et al., 2001) may suggest an increased risk. Given that physical conditioning can attenuate the symptoms of EIMD (McHugh et al., 1999), the potential two months

of 'detraining' during the off-season may render players more susceptible to damage and further injury. In addition, the fact that recovery from pain following EIMD can occur sooner than restoration of neuromuscular performance capabilities (Rinard et al., 2000), may prompt athletes' return to competitive participation possessing potentially reduced levels of dynamic protection.

Figure 1.5. Possible interrelationships between exercise-induced muscle damage (EIMD), neuromuscular performance and ACL injury.



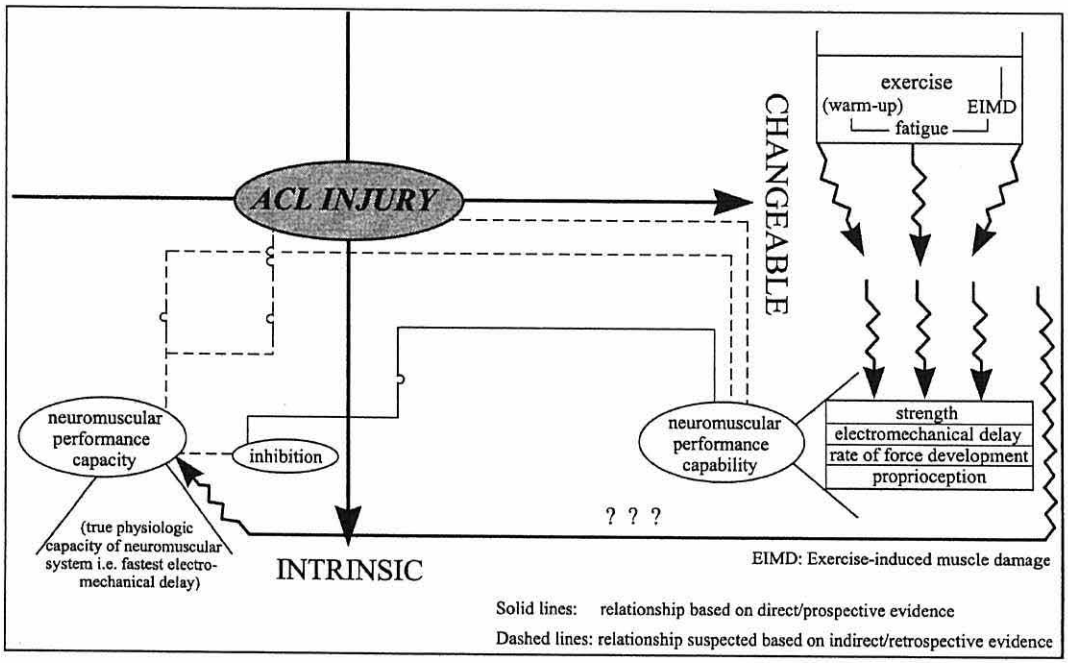
Despite the potential importance of EMD within a conceptual model for knee joint stability, the investigation of such temporal capabilities following this type of exercise is limited. Strojnik and Komi (1998) reported no impairment to electrically evoked EMD following EIMD. However, significant impairments to proprioceptive capabilities have been reported subsequent to eccentric exercise (Saxton et al., 1995). Clearly, further investigation is required given the potential important implications for knee injury, particularly since it seems that the fibres most capable of eliciting timely protective responses (fast twitch) are most

susceptible to damage (Linnamo et al., 2000; Brockett et al., 2001b; Friden and Lieber, 1992).

1.5 *Methods of assessment of neuromuscular performance capabilities*

Traditionally, neuromuscular performance capabilities have been estimated by means of maximal voluntary muscle activation (MVMA). However, the proper evaluation of such performance capabilities can be confounded, consciously or sub-consciously, by waning motivation, injury and associated neuromuscular inhibition (Hopkins and Ingersoll, 2000; Gleeson, 2001). Accordingly, methods of evoked muscle activation have been employed as a means of access to the true (protective) capacity of the neuromuscular system. Conventional electrical stimulation has been the preferred method, however, since this is often painful for the recipient, the painless technique of magnetic stimulation has become more popular (Jalinous, 1995; Barker et al., 1997). Investigation into the effects of exercise on neuromuscular performance capacity is limited to studies utilising electrical stimulation and scrutiny of the results reveal conflicting findings of unchanged (Strojnik and Komi, 1998), impaired (Zhou, 1996) and even improved (Sahlin and Seger, 1995) performance. Given the potential 'pain-free' advantages of magnetic stimulation compared to electrical methods and indeed that the presence of pain can elicit neuromuscular inhibitory mechanisms (Hopkins and Ingersoll, 2000), magnetic stimulation may be an ideal technique by which to assess the true physiologic capacity of the neuromuscular system (King and Chippa, 1989). Such evaluation of performance may be associated with a more accurate estimate of the potential neuromuscular protective capacity and thus enable a more proper establishment of the potential effects of exercise on dynamic protective capacity.

Figure 1.6. Possible interrelationships between neuromuscular performance capability, absolute neuromuscular performance capacity as measured by magnetic stimulation, inhibition and the effects of maximal exercise on ACL injury susceptibility.

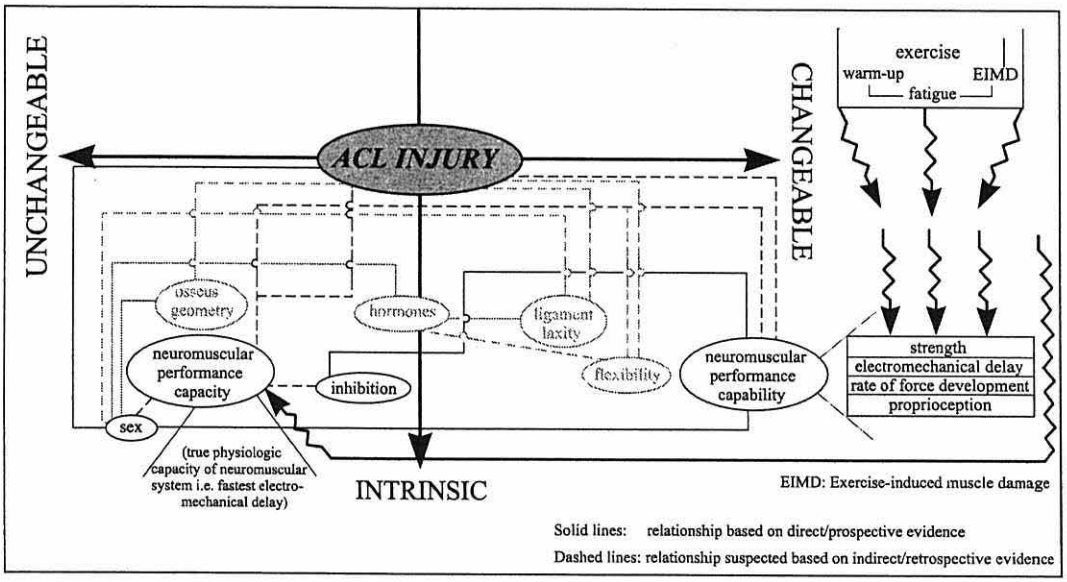


1.6 *The female athlete*

The importance of elucidating fully the processes that may compromise neuromuscular performance may be amplified when considering the dynamic stabilisation of the knee joint in the female athlete. While team sports may represent a hostile environment in which there is generally a substantive threat of ACL injury, research shows that females appear to be at 5 to 8 times greater risk of ACL injury by comparison to male counterparts given equivalent exposure to sport (Gray et al., 1985; Rees, 1994; Arendt and Dick, 1995; Hutchinson and Ireland, 1995; Ireland et al., 1997). The associated mechanisms are currently unclear and are the subject of contemporary debate, although, some researchers have reported a significantly longer EMD in females compared to male counterparts (Bell and Jacobs, 1986; Winter and Brookes, 1991). Increased threat to knee joint integrity is likely to reflect a complex interaction of passive and active structures influenced to

varying degrees by different processes. However, the application of new techniques such as magnetic stimulation may enable the investigation of potential sex-linked differences in temporal neuromuscular capacity and perhaps more importantly, how this capacity is affected by exercise.

Figure 1.7. Possible interrelationships between sex, neuromuscular performance capability, absolute neuromuscular performance capacity as measured by magnetic stimulation, inhibition and the effects of maximal exercise on ACL injury susceptibility.



1.7 *Methods of assessment of neuromuscular performance capabilities; measurement utility*

Given the potential importance of neuromuscular performance capabilities to the capacity to avoid ACL injury, the associated assessment tools should possess adequate precision of measurement that enables confident discrimination of performance differences between, or indeed, within the individual. Accordingly, the reproducibility and reliability characteristics of these indices should be established in order to make meaningful performance comparisons (Mercer and Gleeson, 2002). Currently, however, the inter- and intra-individual measurement

utility of indices of neuromuscular performance is yet to be established for the knee flexors, particularly for the technique of magnetic stimulation.

Chapter 2
Review of Literature

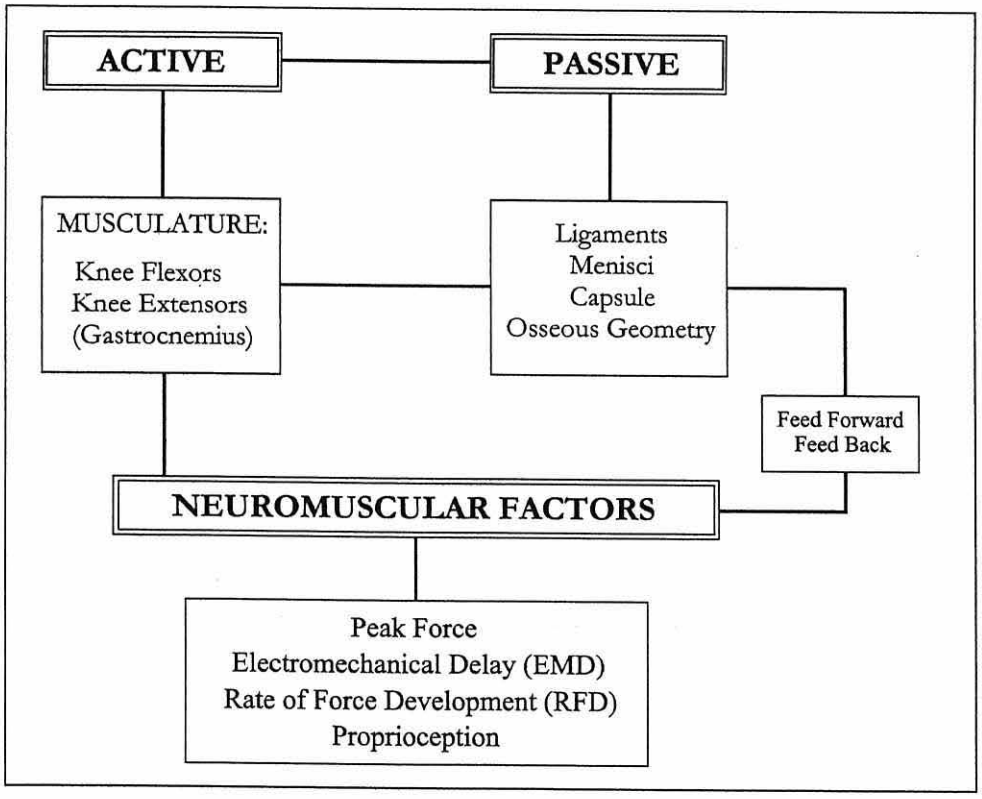
2.0 REVIEW OF LITERATURE

2.1 *Dynamic knee joint stability and anterior cruciate ligament injury*

As highlighted earlier, a conceptual model that defines dynamic knee joint stability depicts the interaction of 'so called' passive structures (osseous geometry, ligaments, menisci and capsular structures) with the active stabilisers (musculature) (Johansson et al., 1991; Fu et al., 1993) (please see figure 2.1). The accumulating evidence of an anterior cruciate ligament (ACL) injury epidemic in team sports athletes (Rees, 1994; Arent and Dick, 1995; Hutchinson and Ireland, 1995; Ireland et al., 1997) estimates the incidence of such injuries to be approximately 30 per 100 000 head of the British population per year and account for approximately 50% of all knee ligamentous injuries (Bollen, 1998). Furthermore, the majority of ACL injuries (estimated 78%) occur by means of non-contact mechanisms (Noyes et al., 1983). These data, considered concomitantly with the fact that untreated, ACL injuries are the most common cause of permanent disability in sport (Kujala et al., 1995), underscore the potential importance of neuromuscular mechanisms in the maintenance of knee joint integrity. Recent evidence also shows females to be at five to eight times greater risk by comparison to male counterparts (Gray et al., 1985; Rees, 1994; Arendt and Dick, 1995; Hutchinson and Ireland, 1995; Ireland et al., 1997). While the mechanisms responsible for this phenomenon remain the subject of contemporary debate, some of the suggested risk factors include sex differences in lower extremity alignment, muscle strength and conditioning of the knee flexors and extensors and exaggerated influences of female hormones on tissue compliance (Hutchinson and Ireland, 1995; Wojtys et al., 1998; Harmon and Ireland, 2000). For example, fluctuations in the laxity of the ACL have been

correlated with concomitant fluctuations in the female sex-hormones progesterone, estradiol and estradiol (Deie et al., 2002; Romani et al., 2003). Furthermore, significant statistical association between stage of the menstrual cycle and the likelihood of ACL injury has been reported (Wojtys et al., 1998; Slauterbeck et al., 2002). Additional risk factors may relate to a prolonged latency of response of the active stabilisers of females by comparison to males counterparts (Bell and Jacobs, 1986; Winter and Brookes, 1991), identified as paramount to the protective capabilities (Gleeson et al., 1998b; Mercer et al., 1998).

Figure 2.1. Simplified conceptual model for knee joint stability.



The predominant non-contact aetiology of ACL injuries highlights the potential importance of neuromuscular protective mechanisms of the active stabilisers. Optimal functioning of the knee flexors is fundamental to the prevention of anterio

cruciate ligament (ACL) injury (Noyes et al., 1986; Rees, 1994; Gleeson and Mercer, 1996), particularly during strenuous activities, where loading of the knee joint can often exceed the strength of the passive structures (Johannson et al., 1991). The knee flexors provide the primary active restraint to tibio-femoral rotation and anterior tibio-femoral translation, the most common mechanisms of ACL injury (Noyes et al, 1983; Rees, 1994; Gleeson et al., 1997). The potential protective capabilities of the active knee stabilisers have been estimated previously on the basis of peak force performance (Arvidsson et al., 1981; Synder-Mackler et al., 1993; Shelbourne and Foulk, 1995), however, the effectiveness of such protective responses may not be solely determined by the absolute strength of the musculature. Contemporary research has indicated that the reaction time of the neuromuscular system to imposed dynamic forces may be equally as important (Wojtys and Huston, 1996), specifically, the index electromechanical delay (Gleeson et al., 1998a; Mercer et al., 1998).

A limited time frame exists whereby potentially harmful dynamic forces must be overcome by the most rapid response of the neuromuscular system in order to protect ligamentous tissue against injury. For the ACL, the time frame from the initial application of such forces to the complete rupture of the of ligament has been estimated at 300 ms (Rees, 1994). However, the reaction time response of the neuromuscular system is limited by a series of processes associated with the production of muscle force (Devita and Skelly, 1992; Gleeson et al., 2000). One aspect of overall neuromuscular reaction time is electromechanical delay (EMD), which represents the time lag between the onset of electrical activity and tension development in skeletal muscle (Norman and Komi, 1979; Zhou et al., 1996). Of

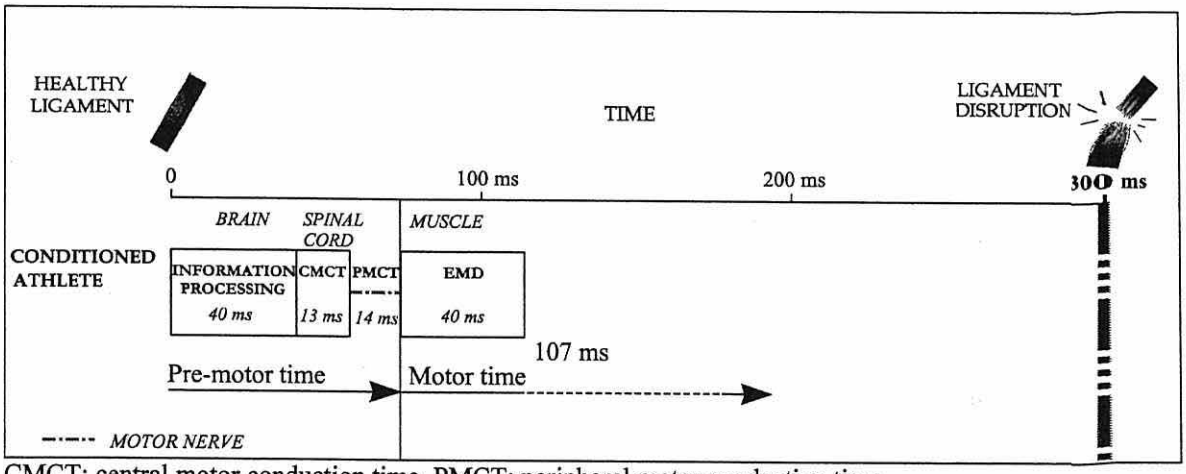
the individual neuromuscular factors involved in the production of meaningful levels of muscle force proposed within the conceptual model for joint stability (figure 2.1), EMD shows the greatest potential for change, demonstrating up to 70% increases (35.3 ± 6.4 ms vs. 60.2 ± 2.0 ms) following acute muscle fatigue (Zhou et al., 1996). This change in performance far exceeds the potential impairments of peak force (53-56% (Zhou et al., 1998; 1996, respectively)), the rate at which muscle force can be mustered (38-53% (Zhou et al., 1996; 1998, respectively)) and proprioceptive capability to detect and respond joint perturbations (~55%, Lattanzio et al., 1997) following similar exercise protocols. Clearly, such temporal impairments coupled with concomitant decreases in the capability to generate peak force may affect substantively the timely harnessing of joint forces of sufficient magnitude to damage ligamentous tissue (Mercer et al., 1998).

2.2 *Components of neurophysiological latency*

In a 'worst case' scenario, the athlete may be required to rely on visual stimuli to cue the initiation of emergency injury avoidance responses. Compared to proprioceptive sensations at the involved joint, neuromuscular responses to visual stimuli may be associated with a longer time delay (Pope, 1979) possibly due to the required information processing (IP) by the brain and a potentially greater distance of neural propagation to the target musculature. Accordingly, such reactions involve: IP, transmission of the generated 'response' from the brain to the appropriate site on the spinal cord (central motor conduction time), propagation of the action potential from the spinal cord to the target musculature (peripheral motor conduction time) (Benecke, 1996) and EMD. Electromechanical delay alone may account for a substantive portion of the entire time delay associated with the

initiation of muscle force as the following calculations and schematic (figure 2.2) will demonstrate. Collectively referred to as pre-motor time (Yeung et al., 1999), the expected latency from IP to the end of peripheral motor conduction time (PMCT), based on published values, may approximate 67 ms (please see figure 2.2) (information processing: 30-40 ms (Brebner and Welford, 1980), central motor conduction time (CMCT): 13 ms and PMCT: 14 ms to the knee extensors (Ugawa et al., 1989)). Initiation of muscle force is then subject to a electromechanical delay (detailed discussion in sub-chapter 2.3) which may approximate 40 ms (Zhou et al., 1996, data for the knee extensors). Figure 2.2 shows that in response to visual cues, a predicted 107 ms may elapse prior to the production of discernible levels of muscle force. This temporal delay accounts for one third of the total time threshold for ACL injury, of which EMD may occupy approximately 37%.

Figure 2.2. Latency of dynamic muscle response.

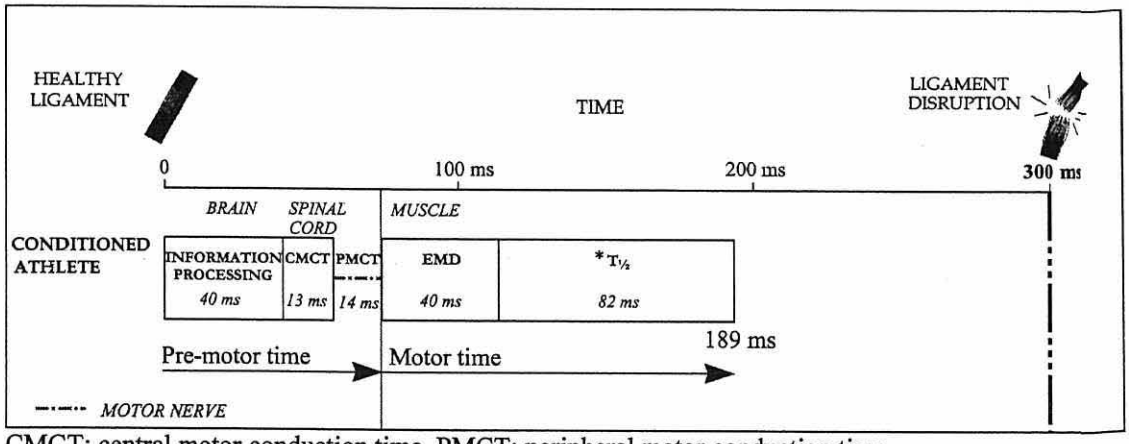


CMCT: central motor conduction time, PMCT: peripheral motor conduction time
EMD: electromechanical delay.

In order to extrapolate such performance values to emergency scenarios and estimate potential injury susceptibility, it is appropriate to include the expected latency associated with the generation of meaningful levels of force. This may be

estimated arbitrarily on the basis of time to half peak force ($T_{½}$). Absolute temporal scores for $T_{½}$ may be expected to vary according to peak force capabilities, however, values have been reported to range from 70 to 100 ms (unpublished data: Gleeson, personal communication) for the knee flexors and 116 to 126 ms for the knee extensors (Hakkinen and Komi, 1983; Kyrolainen and Komi, 1994) for the healthy individual. While figure 2.3 represents a fairly crude series of calculations, the diagram shows that under un-fatigued conditions, the healthy athlete might be expected to possess the necessary neuromuscular performance capabilities to maintain dynamic stability of the knee joint in response to mechanical loading. Figure 2.3 also illustrates that approximately 20% of the total expected latency of muscle response is accounted for by EMD alone. Given that this index of temporal neuromuscular performance may be substantively prolonged following muscle fatigue (Horita and Ishiko, 1986; Zhou et al., 1996; Gleeson et al., 1997, 1998b), such may further emphasise the potentially integral role of EMD in defining the capability to resist knee joint injury. These issues will be addressed in detail later (please see section 2.6.2) following the examination of the index of EMD.

Figure 2.3. Total latency of dynamic muscle response.



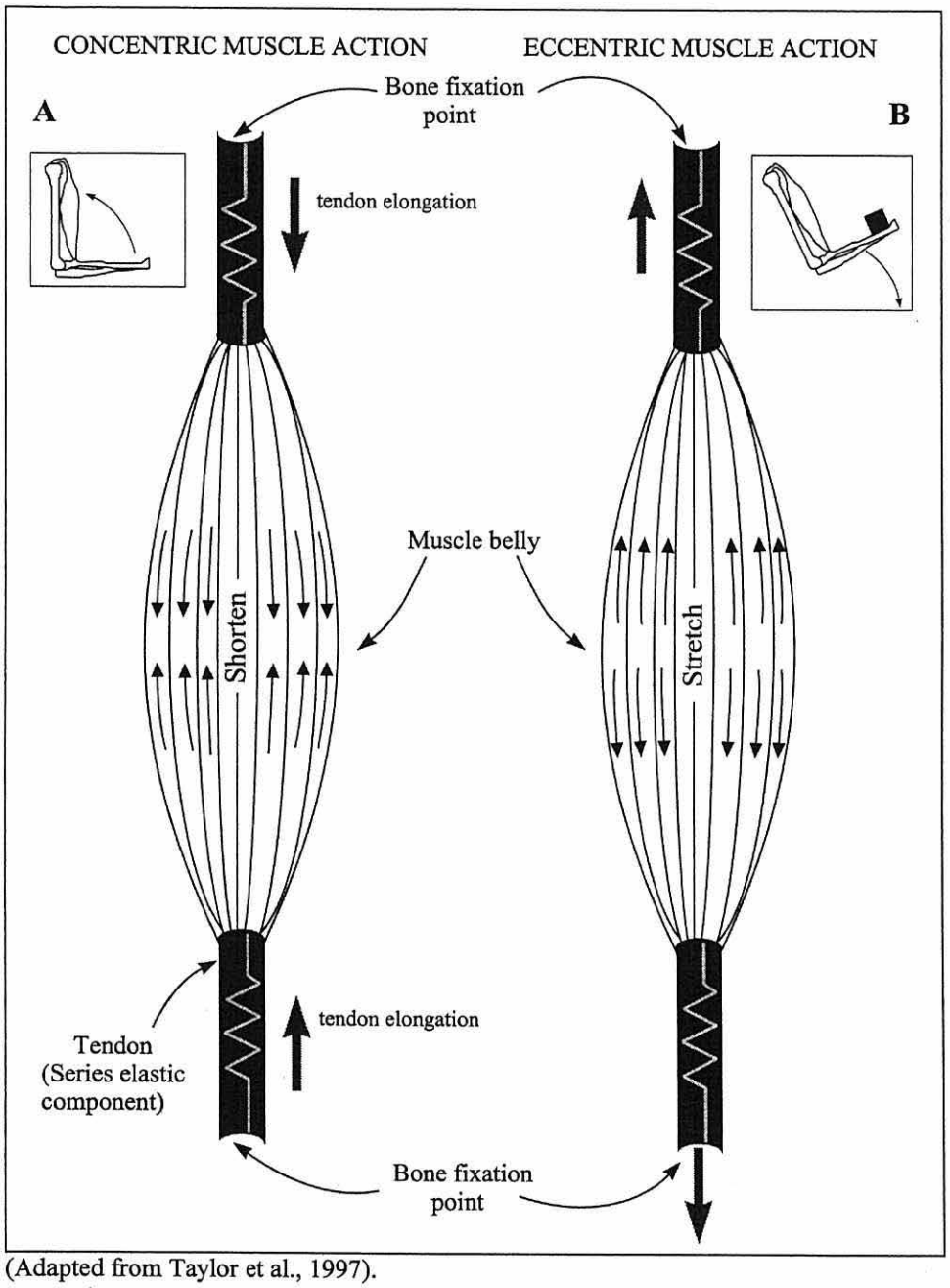
CMCT: central motor conduction time, PMCT: peripheral motor conduction time
EMD: electromechanical delay, $T_{½}$: time to half peak force.
*$T_{½}$ value selected arbitrarily from lower range reported for the knee flexors to represent likely values for the conditioned athlete.

2.3 *Components of electromechanical delay (EMD)*

Electromechanical delay is comprised of several events associated with excitation contraction coupling and as such, can offer a potentially important insight into the neuromuscular and musculoskeletal performance of a joint system (Gleeson, 2001). Defined as the time between the onset of muscle electrical activity and the onset of force (Norman and Komi, 1979; Zhou et al., 1996), the processes of EMD include: conduction of the action potential through the muscle and the t-tubule system, release of calcium (Ca^{2+}) ions from the sarcoplasmic reticulum, formation of the actin-myosin cross-bridges and the lengthening of the series elastic component (SEC) (Cavanagh and Komi, 1979; Komi, 1979). Despite these many mechanisms, research shows that muscle fibre conduction velocity (MFCV) may only account for a small portion (approximately 22%) of the total delay (Zhou et al., 1998), whereas the stretch of the SEC by the contractile component may explain the majority of the EMD (Cavanagh and Komi, 1979; Komi, 1979; Norman and Komi, 1979 Zhou et al., 1998; Granata et al., 2000; Kubo et al., 2000). The predominant influence of the SEC on EMD performance was initially proposed by early studies focussing on EMD differences subsequent to eccentric compared to static and/or concentric muscle actions (Cavanagh and Komi, 1979; Norman and Komi, 1979) and was more recently confirmed by investigations into the effects of clinical muscle spasticity (Granata et al., 2000) and the elastic properties of tendon structures following bed rest (Kubo et al., 2000). In all cases, EMD values were significantly shorter where the SEC was already under stretch (eccentric vs. concentric/static actions; spastic muscle vs. healthy control), or in the case of bed rest, where the SEC was comprised of stiffer tissue (pre- vs. post-bed rest). The influence of the SEC, which is comprised primarily of the tendon and the connective tissue layers

(McComas, 1996), manifests during muscle activation by acting like a spring resisting external attempts to change muscle length (Latash, 1998). Consequently, the registration of muscle force will occur only when the SEC has been stretched (Komi, 1979). The latency associated with the elongation of the elastic component can vary according to the conditions of muscle activation. For example, the typically shorter EMD recorded during eccentric compared to static and concentric muscle actions, is thought to be due to the lengthening of the muscle adding to the velocity of the stretch of the SEC (Cavanagh and Komi, 1979; Norman and Komi, 1979). Under concentric or static conditions, however, the SEC must be elongated as the muscle fibres shorten prior to the registration of muscle force (please see figure 2.4).

Figure 2.4. Muscle-tendon unit undergoing concentric(/static) and eccentric actions.



(Adapted from Taylor et al., 1997).
A: Tendon must be stretched during muscle shortening to bring about movement (the SEC also undergoes stretch during static muscle actions).
B: Stretch of the muscle adds to the stretch of the SEC as force is developed.

The duration of the recorded EMD can also be influenced by the definition adopted to describe the latency and the threshold value chosen to identify the end point of the delay. For example, the onset of acceleration or movement (e.g. Norman and

Komi, 1979; Gleeson et al., 1998b) will clearly yield a longer EMD by comparison to where the onset of muscle force (e.g. Zhou et al., 1996; Chan, et al., 2001) is the selected end threshold. The following sections will explore how such factors may impact EMD.

2.4 *Electromechanical delay calculation and measurement*

2.4.1 *Methods of electromechanical delay calculation*

Identification of the precise time at which the muscle electrical activity and force begins is a challenging task for the experimenter, particularly considering that these data can be confounded by technical and biological variability (Gleeson, 2001). Two key approaches to calculating the latency associated with EMD have been developed, these include visual inspection of the data records and the cross-correlation technique. The simplest approach involves the visual inspection of the computer data records by means of appropriate software to discriminate the recorded EMG signal and muscle force from background noise. Criterion thresholds for force generation, which should be set to exceed the technical error of the recording system, have been previously utilised, for example an absolute Newton value (1N: Kyrolainen and Komi, 1994), or a percentage value of peak force (5% peak force: Sahlin and Seger, 1995). Further scientific rigour can be achieved by the construction of 95% confidence limits around the mean noise amplitude, enabling a statistically sound detection of EMG and force onset (Gleeson, 2001). The initiation of activation of the muscle is noted when the respective signals exceed the 95% confidence limits for a pre-defined minimal period (Gabriel and Boucher, 1998; Gleeson, 2001). A disadvantage of the 'visual

inspection' method is the potential for subjective error, however such may be minimised by the corroboration of data interpretation by a second experimenter. In addition, research shows reproducibility characteristics (coefficient of variation (V%)) associated with this method of EMD calculation (V%: 6.6% (Gleeson et al., 1998)) that are comparable to other indices of neuromuscular performance, such as static peak force (4.1% (Viitasalo et al., 1980)).

A second method of estimating EMD performance is by means of cross-correlation techniques, where a linear envelope without phase shift is constructed with respect to the raw, rectified EMG signal. The temporal shift between the linear envelope and the force recorded is established by cross-correlation procedures, where the EMD is defined as the delay where the highest correlation is observed (Vos et al., 1990). This method of estimating EMD performance appears to be the preferred approach subsequent to sub-maximal muscle actions, where muscle activation characteristics are externally cued through 'ramp and hold' mechanisms (Vint et al., 2000), or guided by metronome (Vos et al., 1990; van Dieen et al., 1991; Vos et al., 1991). While cued muscle actions ensure a smooth and regulated activation and relaxation pattern, enabling an average temporal shift across a series of replicates to be calculated, it also prevents the accurate determination of the extent of agreement between the two different methods in the estimation of EMD performance capabilities. This is because studies employing the visual method have estimated EMD performance subsequent to maximal, explosive muscle actions (e.g. Zhou et al., 1996; Paasuke et al., 1999; Chan et al., 2001). While an advantage of cross-correlation could be the obtaining of a global measure of phase shift which may then be applied to a series of kinematic data (De Luca, 1997), the conditions under

which these estimates of EMD performance are acquired are often not representative of the individual's true performance capabilities. In order to extrapolate the recorded data to a selected performance situation, the experimental setting should replicate as much as possible the desired scenario. In an emergency scenario where imposed joint loads must be restrained by the most rapid response of the neuromuscular system (Swanik et al., 1997), recruitment from the pool of large high threshold fast motor units is likely to be substantive in an attempt to preserve joint integrity. Such motor unit strategies are unlikely to be elicited subsequent to relatively slow sub-maximal conditions (Garnett and Stephens, 1981). Consequently, the ultimate performance capabilities of the neuromuscular system should be estimated subsequent to maximal activation of muscle, particularly when the data obtained may play an integral role in the clinical judgement of likely dynamic protective capacity.

The method of visual inspection of each data record appears to be the simplest and most widely utilised technique for calculating EMD. The technique of cross-correlation may require the regulation of muscle activation and relaxation and as such, may not be the preferred method to calculate EMD during momentary maximal explosive muscle performance assessments. However, if it can be assumed that the force produced is from the measured muscle and there is no antagonistic coactivation (Vos et al., 1990), cross-correlation may prove effective where stable baseline values cannot be discerned and/or during dynamic muscle actions. Bi-directional isokinetic dynamometry may be one such application (Gleeson, 2001).

2.4.2 *Methods of muscle activation and measurement of electromechanical delay*

Several factors can potentially confound the proper measurement of an individual's true maximal neuromuscular performance capacity under volitional conditions, either consciously or sub-consciously, such as waning motivation, injury and associated neuromuscular inhibition (Hopkins and Ingersoll, 2000; Gleeson, 2001). Accordingly, methods of evoked muscle activation have been employed as a means of access to this maximal capacity (King and Chippa, 1989). Traditionally, electrical stimulation (ES) has been the preferred method, both in assessment (Behm and Sale, 1994; Behm and St-Pierre, 1997) and rehabilitation (Morrissey et al., 1985; Dilitto et al., 1988; Snyder-Mackler et al., 1994) settings, however, ES is typically painful for the recipient (Jalinous, 1995). More recently, the technique of magnetic stimulation (MS) has become increasingly used. The progress of MS as a means of investigating neuromuscular performance has been rapid since the achievement of MS of the human motor cortex by Barker and colleagues in 1985. Magnetic stimulation delivers a brief intense magnetic field that causes depolarisation of the underlying tissue (Benecke, 1996). Developments in the design of stimulating coils which deliver the magnetic pulse to the target area, have made possible the focal stimulation of the motor cortex and deep peripheral motor nerves (Jalinous, 1995). The primary advantage of MS over conventional electrical stimulation is that while ES is typically painful to the recipient, MS is characterised by the absence of a pain response (Jalinous, 1995; Benecke, 1996). Unlike ES, MS is able to penetrate body structures without attenuation, in addition, the decrease in induced electrical field with distance into the body from a magnetic coil is much less than that due to currents applied to surface electrodes (Barker, 1987). Accordingly, MS is able to penetrate considerable depths without causing electrical

fields (and pain) at the surface (Barker, 1987). A further advantage of MS is that direct contact of the magnetic stimulating coil with the body is not necessary, whereas ES requires preparation of the skin over the site of the stimulating electrodes (Benecke, 1996). Clearly, in situations where cutaneous injury might be present, MS possesses advantages over ES. Positioning of the magnetic coil against the skin, however, can be an effective method to standardise the location (e.g. Polkey et al., 1996). Criticisms of MS include the difficulty in obtaining supramaximal stimulation (Evans et al., 1988; Britton et al., 1990), defined as a plateau of the amplitude of muscle response despite increases in stimulation intensity (King and Chippa, 1989). Supramaximal stimulation is desirable to guarantee stimulation of the fastest conducting fibres, although it has been stated that this should not be necessary, since the fastest conducting fibres should have the lowest threshold to MS and therefore, be activated even with sub-maximal intensities (King and Chippa, 1989). Several studies have, however, achieved supramaximal stimulation by means of magnetic stimulation (Cross et al., 1990; Polkey et al., 1996; Hollge et al., 1997), possibly as a result of the progressive development of magnetic coils, enabling the peak magnetic field to be more accurately focussed on the chosen nerve (Jalinous, 1995).

Magnetic stimulation of a motor nerve is associated with the capability to activate the fastest motor units (Maertens de Noordhout, 1991) and given the factors that might potentially confound the proper measurement of an individual's true maximal performance capacity (Hopkins and Ingersoll, 2000; Gleeson, 2001), this technique is an ideal method of offering an insight into the true maximal physiological performance capacity of the neuromuscular system (King and Chippa, 1989).

Several studies have utilised MS to investigate peripheral and central conduction time latencies (e.g. Britton et al., 1990; Hollge et al., 1997). However, there has been limited scrutiny into the temporal capacity to initiate muscle force by this method. Those studies that have investigated EMD responses subsequent to methods of evoked muscle activation have utilised electrical stimulation (e.g. Tsuji and Nakumura, 1988; Sahlin and Seger, 1995; Zhou et al., 1995; Zhou, 1996; Strojnik and Komi, 2000; Kaneko et al., 2002). These results show that EMD responses subsequent to electrical stimulation are typically quicker than elicited by means of MVMA, possibly confirming the existence of potential inhibitory mechanisms which may limit volitional access to fast twitch motor units. For example Zhou et al. (1995) recorded an EMD of 38.7 (± 5.4) ms of the knee extensors during MVMA, which was significantly longer than elicited by electrical stimulation (17.2 ± 2.8 ms). While direct comparisons of temporal responses evoked by means of ES and MS cannot be made, MS appears to elicit similar force responses to ES. Polkey et al. (1996) recorded a group mean peak twitch force response of the knee extensors of 120 (± 31.4) N subsequent to a single supramaximal magnetic stimulus of the femoral nerve, representing approximately 17% of MVMA. This compares to the approximate 15% of MVMA (86.2 N (no SD reported) of the same muscle group observed by Zhou et al. (1995) elicited by means of electrical stimulation (150V) over the muscle belly. Based on the suggestion of similar muscle activation characteristics to ES and the absence of a pain response, MS may be a preferred method of assessment of performance capacity over ES. Evaluation of performance capacity by means of MS would minimise participant discomfort and reduce the likelihood of potential pain-related

neuromuscular inhibitory mechanisms that may be associated with ES (Hopkins and Ingersoll, 2000).

2.5 *Measurement utility of electromechanical delay*

The utility of EMD as an index of neuromuscular performance has been questioned by some researchers (Grabiner, 1986; Bochdansky et al., 2001), primarily due to the wide range of absolute values reported in the literature, even for the same muscle (e.g.: 38.0 ms to 106.0 ms for the rectus femoris (Zhou et al., 1996; Vos et al., 1991), respectively). Varying absolute performance scores, however, should not necessarily be the sole criterion by which to judge the utility of this parameter, particularly since diverse methodologies may have differentially influenced the magnitude of EMD scores. As mentioned previously, specific interventions and factors such as joint angle, type and level of muscle activation and EMD definition can affect absolute performance scores and are likely to account for a substantive portion of the variation in EMD values between studies. For example, both Zhou et al. (1996) and Vos et al. (1991) followed synonymous protocols for joint angle (90° knee flexion) and type of activation of the knee extensors (static), however the EMD of 106.0 (± 16.5) ms recorded by Vos et al. (1991) was subsequent to 50% MVMA, compared to the 38.0 (± 8.1) ms during maximal muscle activation reported by Zhou et al. (1996). These data are consistent with the tenet that greater proportions of high threshold fast motor units, associated with a quicker EMD due to faster more powerful contractile capabilities compared to other motor units (McComas, 1996), are recruited during maximal compared to sub-maximal conditions (Garnett and Stephens, 1981). Comparison of the studies by Morris and Beaudet (1980) and Bell and Jacobs (1986) also reveal very different EMD values

for the same muscle, however, differences in measurement resolution may be a potential source of variation between these investigations. For example, Morris and Beaudet (1980) recorded an EMD for the biceps brachii 51.5 ms (no SD reported) subsequent to maximal static muscle activation of the elbow flexors, whereby immediate force onset was reliant on the deflection of a marker pen on a paper chart recorder set at a speed of 200 $mm.s^{-1}$. The much lower EMD values recorded by Bell and Jacobs (1986) (34.5 (±16.3) ms to 25.1 (±10.4) ms), despite following the same procedures, may be a consequence of high speed analogue to digital sampling (2000 Hz) which may enable a more consistently accurate detection of force onset (5 N). The expected measurement utility of EMD should, therefore, be evaluated by means of examining the associated reproducibility and reliability characteristics (Gleeson and Mercer, 1996), particularly since many principle sources of inter-investigation variation in absolute EMD performance can be experimentally controlled.

During inter-group comparisons, a desired level of experimental power may be achieved through manipulation of the number of participants within each sub-sample, given the expected sample means and standard deviations (Lipsey, 1990). However, case-study investigations demand stringent criteria for precision of measurement in order to make meaningful performance comparisons (Mercer and Gleeson, 2002). The minimally acceptable level of measurement sensitivity may be dependant on the assessment setting, however, the selected index (indices) should be sensitive to small changes in performance capability during the evaluation of performance of the elite athlete or a patient population approaching optimally rehabilitated status (Gleeson and Mercer, 1992). For example, the performance

levels of the elite strength athlete may be expected to vary by only ±5% over the competitive season (Gleeson and Mercer, 1992). Investigations into the performance variability of EMD of the knee flexors and extensors have reported intra-day coefficients of variation (V%) of 6.1% (Gleeson et al., 1998) and 8.2% (Viitasalo et al., 1980), respectively. These V% scores generally indicate slightly higher variability of performance based on a single trial by comparison of indices of isokinetic knee flexor (e.g. 4.9% (Gleeson and Mercer, 1992)) and static knee extensor (e.g. 4.1% (Viitasalo et al., 1980)) peak force, respectively. While these results do not advocate the utilisation of single trial EMD protocols, these data show that this index offers equivocal, if not superior, measurement precision by comparison to other commonly used EMG variables such as integrated EMG (V%: 6.9%) pre motor time (18.8%) and total reaction time (16.5%) (Viitasalo et al., 1980).

Reports of good intra-day and inter-day reliability of EMD performance (R_I = 0.93 (Viitasalo et al., 1980) and 0.95 (Klimovitch, 1977), respectively) suggest measurement sensitivity characteristics that enables discrimination of the performance capabilities of particular individuals from within the group (Gleeson and Mercer, 1996). However, in order to make confidently intra-group comparisons, concomitant consideration should be given to the performance heterogeneity of the group, since such can falsely inflate the reliability coefficient (Hopkins, 2000). Currently these aspects of EMD measurement sensitivity are yet to be established. Reliability characteristics of an index of performance that can enable the separation of individuals from a group may be vital in scenarios where resources are limited. For example, identification of individuals who require further

treatment from a group of patients is likely to be a key aim in contemporary clinical practice.

While EMD may be associated with superior measurement sensitivity characteristics to other EMG indices, further investigation into the reproducibility and reliability characteristics of this index is required to more accurately determine the likely measurement precision afforded during case study investigations.

2.6 *Factors influencing electromechanical delay*

As mentioned previously, several factors can potentially impact the recorded EMD, however, many of these can be experimentally managed and manipulated. These will be discussed in the proceeding sub-chapters according to their classification of being intrinsic (uncontrollable) or extrinsic (controllable).

2.6.1 *Intrinsic*

Sex-linked differences and electromechanical delay

The initial processes involved in the conversion of excitation into muscle force comprise a relatively small portion of the entire electromechanical delay compared to the stretch of the SEC (Cavanagh and Komi, 1979; Norman and Komi, 1979; Komi, 1979; Winter and Brookes, 1991; Zhou et al., 1998; Kubo et al., 2000). The influence of the SEC is thought to be the principle mechanism where differences in EMD performance are observed between males and females (Bell and Jacobs, 1986; Winter and Brookes, 1991), since the influence of female specific hormones is

believed to induce greater compliance in the SEC (Wojtys et al., 1998; Harmon and Ireland, 2000). Yet, the existence of such sex-linked EMD performance differences is debated in the literature, with equal numbers of studies reporting parity (Morris and Beaudet, 1980; Houston et al., 1988) and systematic differences (Bell and Jacobs, 1986; Winter and Brookes, 1991) between males and females. For example, Winter and Brookes (1991) reported significantly longer EMD values of the soleus muscle in female participants (44.9 ± 6.6 ms) by comparison to males (36.9 ± 4.0 ms). They concluded that because females also possessed a longer elastic charge time (time between the registration of muscle force and the onset of movement) compared to males, musculotendinous elasticity accounted for at least part of the longer EMD. These findings are in agreement with an earlier study reporting significantly longer EMD performance scores for females compared to male counterparts (Bell and Jacobs, 1986). Bell and Jacobs, (1986) retrospectively sub-divided participants according to absolute strength performance of the biceps brachii within each sex group, based on the tenet that greater force generating capabilities may predispose a quicker stretch of the SEC compared to weaker muscles. While both strong and weak males possessed a significantly quicker EMD (25.1 (± 10.4) ms, 26.7 (± 10.3) ms, respectively) by comparison to both females groups (strong females 32.2 (± 12.8) ms, weak females 34.5 (± 16.3) ms), the absolute strength comparisons between sexes were not equivalent. Since weak males were significantly stronger than strong females, the sex-linked differences in EMD performance reported by Bell and Jacobs (1986) may also represent potential strength and/or muscle fibre composition differences between males and females. However, the significant difference in force generating capabilities between strong and weak participants within the same sex groups, yet equivalent EMD scores

suggests factors other than strength performance capabilities may have greater influences on temporal neuromuscular performance. Contrary to the aforementioned studies, Morris and Beaudet (1980) and Houston et al. (1988) did not observe significant EMD differences between males and females. Houston et al. (1988) reported statistically similar EMD performances and also muscle fibre composition between the sexes in the vastus lateralis. Morris and Beaudet (1980) observed parity in EMD performance in muscles of the finger (flexor digitorum fundus), knee (rectus femoris), wrist (flexor digitorum superficialis), elbow (biceps brachii) and ankle (tibialis anterior) between males and females. In the absence of further research on muscle composition and the compliance characteristics of the SEC, conclusions cannot be currently drawn regarding potential EMD performance differences between sexes. However, it is interesting to note that the studies reporting no sex-linked differences in EMD performance relied on analogue recording and interpretation of data, in contrast to Bell and Jacobs (1986) and Winter and Brookes (1991). Interrogation of the data records by computer software and high speed analogue to digital sampling employed by Winter and Brookes (1991) and Bell and Jacobs (1986) (2000 Hz and 2500 Hz, respectively) may have been associated with greater consistency and accuracy in the detection of EMG and force onset by comparison to the determination of movement of a marker pen at a paper speed of 200 $mm.s^{-1}$ and 380 $mm.s^{-1}$ (Morris and Beaudet, 1980; Houston et al., 1988). Such possible minimisation of technical error by contemporary digital techniques may accordingly explain why seemingly larger differences in absolute group mean performance between males and females (approximately 9 ms, (M: 59.6 ± 8.1 ms, F: 68.7 ± 7.7 ms)) were non-significant (Morris and Beaudet, 1980)

compared to smaller performance contrasts (approximately 5 ms M: 39.6 ± 4.0 ms, F: 44.9 ± 6.6 ms) (Winter and Brookes, 1991).

Further research is clearly required to investigate potential sex-linked EMD performance differences. However, females are still likely to contend with similar types of physical and mechanical stresses during sports participation compared to male counterparts. As such, investigation into the potential factors that might induce change in temporal neuromuscular performance may be more effective in the research of injury susceptibility.

Muscle fibre-type and conditions of muscle activation

In addition to the potential influence of intrinsic factors, research shows that the conditions of muscle activation can affect EMD, in particular, the recorded latency has been reported to vary by level of effort (Norman and Komi, 1979; Grabiner, 1986; Vos et al., 1991), type of muscle activation (Cavanagh and Komi, 1979; Norman and Komi, 1979), joint angle (Grabiner, 1986; Vos et al., 1991; Chan et al., 2001) and muscle fatigue (Horita and Ishiko, 1987; Zhou et al., 1996, 1998; Gleeson et al., 1998a; Mercer et al., 1998).

Electromechanical delay is generally accepted to be shorter with increasing levels of muscular effort (Vos et al., 1991; Zhou et al., 1995). For example, Vos et al. (1991) recorded a significantly shorter EMD of the knee extensors during 30% static MVMA by comparison to 50% MVMA (106 ± 16.5 ms vs. 93 ± 12.1 ms). In addition, Zhou et al. (1995) observed a significantly shorter EMD also of the knee extensors during static MVMA compared to 30% MVMA (42.6 ± 8.3 ms vs. 38.7 ±

5.4 ms). Recruitment of a greater proportion of large high threshold fast twitch motor units with increasing levels of muscle activation, which are associated with faster propagation speeds, quicker cross-bridge cycling (McComas, 1996) and potentially faster subsequent stretching of the SEC (Komi, 1979) compared to slow motor units, may be the primary mechanism mediating these differences in performance. Indeed, quicker EMDs have been reported of muscles with greater proportions of fast twitch muscle fibres. For example, Norman and Komi (1979) reported the EMD of the 'fast' triceps brachii to be considerably shorter compared to the biceps brachii comprised of a greater proportion of slow twitch motor units (26 ± 10.5 ms vs. 41 ± 13.1 ms). However, comparisons between strength and power athletes and endurance and untrained individuals, where inter-group proportions of fast twitch muscle fibres might be expected to differ substantively have yielded unequivocal results. Despite significant strength differences between power and endurance athletes (Kyrolainen and Komi, 1994) and strength and untrained groups (Hakkinen and Komi, 1983), EMD scores evoked by means of patella tendon tap reflexes were reportedly similar between differently trained groups. While these results may demonstrate the potential independence of EMD from muscle fibre type composition, the monosynaptic stretch reflex technique used to evoke the EMD responses is controlled by slow motor units (Viitasalo and Komi, 1981) and as such, may not be the most sensitive method by which to assess temporal neuromuscular capacity.

As alluded to earlier, conditions of eccentric muscle activation are typically associated with shorter EMD values compared to static or concentric muscle actions (Norman and Komi, 1979; Cavanagh and Komi, 1979). Cavanagh and Komi (1979)

observed a significantly shorter EMD in the elbow flexors during isokinetic eccentric activation of muscle (49.0 ms) compared to concentric (55.4 ms) and static (53.9) actions (no SD reported). Similarly, Norman and Komi (1979) reported a 13 ms difference in the EMD of the biceps brachii subsequent to eccentric (28 ± 10 ms), compared to concentric muscle activation (41 ± 13 ms). Both studies proposed that the majority of EMD is mediated by the stretch of the SEC, whereby lengthening of the investigated muscle (during eccentric activation) can add to the stretch of the SEC resulting in a shorter EMD. Vos et al. (1991), however, reported a significantly longer EMD subsequent to maximal eccentric compared to concentric isokinetic activation of the knee extensors (104 ± 9.8 ms vs. 90 ± 6.8 ms) at an angular velocity of $60°.s^{-1}$ ($1.04\ rad.s^{-1}$). While the authors did not speculate regarding potential mechanisms, the velocity of muscle actions may have been a contributory factor. Norman and Komi (1979) showed the significantly faster EMDs under eccentric compared to concentric conditions were present only at the fastest movement velocities. The fastest movement velocity employed by Vos et al. (1991) ($1.04\ rad.s^{-1}$) was considerably slower than the movement speeds of Norman and Komi (1979) (approximately $500°.s^{-1}$, ($8.73\ rad.s^{-1}$) and $800°.s^{-1}$ ($13.96\ rad.s^{-1}$)). Potentially greater contributions from the fastest motor units may be expected during the quickest movement velocities (Komi, 1979). In addition, during slow movement velocities, the possible disinhibition of the antagonist muscle caused by the muscle stretch (Latash, 1999) may oppose the response of the agonist and subsequently influence the temporal delay.

Given that the properties of the SEC may directly influence the recorded EMD (Kubo et al., 2000), manipulation of joint assessment angles may induce muscle-

tendon stretch and effect a shorter EMD. Chan et al. (2001) found that the EMD of the vastus lateralis (VL) and medialis oblique was significantly longer the larger the knee flexion angle (175°, 150° and 90° knee extension, 51.2 ± 19.6 ms, 37.7 ± 7.2 ms and 32.1 ± 9.5 ms, respectively for VL [180° = full extension]). In addition, Grabiner (1986) recorded an EMD for the triceps brachii of 47 (± 11) ms subsequent to maximal effort unloaded elbow extensions at 120° elbow flexion, which was significantly shorter than recorded at 100° (52 ± 10 ms) and 80° (57 ± 13 ms) (0° = full extension) of elbow flexion. Interestingly, such EMD differences are not observed under sub-maximal conditions. Grabiner (1986) reported no differences in EMD performance between the three elbow flexion angles (80°, 100° and 120°) during sub-maximal movement velocities (25%, 15% and 10% of minimal movement time). In addition, Vos et al. (1991) did not observe systematic variations in EMD performance by joint extension angle during sub-maximal (50% and 70% MVMA) static activation of the knee extensors (90° and 130° knee extension [180° = full extension]). While certain joint configurations may effect a greater residual stretch of the SEC and the potential for a quicker EMD, such may have minimal influence during sub-maximal conditions, where there is the possibility of reduced contribution from fast high threshold motor units (Garnett and Stephens, 1981). This may be particularly true if such angles also have an unfavourable effect of the degree of overlap of the contractile proteins (length-tension relationship) (McComas, 1996).

The preceding sections have outlined several factors that can influence EMD performance, however, these factors can be managed and manipulated by the experimental protocol and as such, do not necessarily challenge the potential utility

of this index. Rather, these data propose the detailed consideration of the measurement conditions during protocol development to reflect as much as possible the desired 'real-life' performance scenario. The following section will consider the effects of exercise on EMD performance.

2.6.2 *Extrinsic*

Effects of exercise

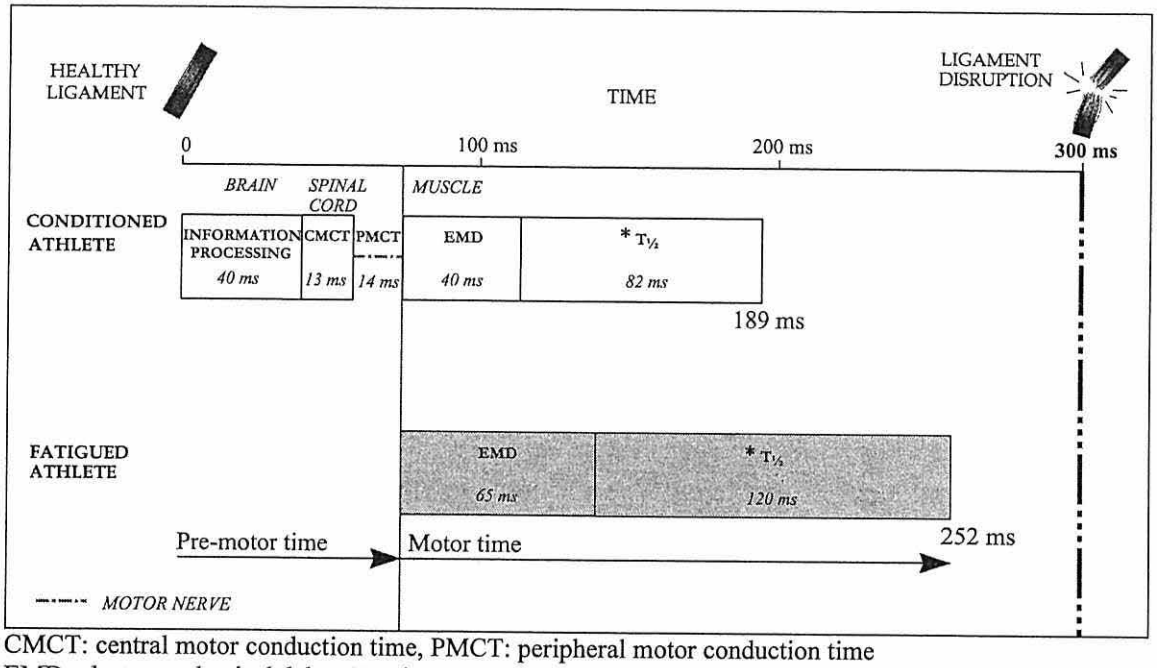
Of all the possible involved processes associated with the initiation of muscle force in response to visual stimuli (detailed in 2.2), EMD appears to be most affected by fatigue. Increases of between 42% to 70% compared to pre-exercise values have been observed subsequent to protocols of MVMA (Horita and Ishiko, 1987; Zhou et al., 1996). Conversely, high intensity exercise seems to have minimal effects on pre-motor processes. For example, Hollge et al. (1997) demonstrated that neither aerobic (50 minutes jogging [8-10 km]) or high intensity anaerobic (400 metre sprint [50-79 seconds], stepping exercise of 600 steps [6-9 minutes]) exercise was associated with impairments to the CMCT or PMCT of the lower extremities (abductor hallucis, tibialis anterior and knee extensors). In fact PMCT demonstrated a slight improvement subsequent to the aerobic and stepping exercise, which was attributed to the increase in conduction velocity associated with exercise-induced temperature increases (Hollge et al., 1997). Potential facilitatory effects of temperature on MFCV and subsequently EMD performance might also be expected, however, research shows that this may not occur when accompanied by fatigue. Zhou et al. (1998) reported that MFCV, in addition to EMD latencies, were generally quicker at high, by comparison to lower, muscle temperatures (range: 30-

38°C) when passively changed, though muscle temperature increases observed subsequent to 25 fatiguing maximal static knee extensions (33.8°C at rest to 35.9°C following exercise), were actually associated with impaired MFCV and EMD performance (15.8% and 45.1% increase, respectively).

As mentioned previously, the protective capability of the active muscle stabilisers to avoid knee injury may be associated substantively with the reaction time of the neuromuscular system to imposed dynamic forces (Wojtys and Huston, 1996), in particular EMD (Gleeson et al., 1997; Gleeson et al., 1998a; Mercer et al., 1998; Gleeson et al., 2000). High intensity exercise may, therefore, present a realistic challenge to the capabilities of the dynamic stabilisers to maintain joint integrity during mechanical loading. Subsequent to a single acute episode of maximal exercise Yeung et al. (1999) reported a 40.4% increase in EMD (39.6 ± 9.0 ms vs. 51.8 ± 1.6 ms) of the vastus medialis and a 15% decrease in peak force following an intermittent protocol (5s MVMA, 5s rest) totalling 30 seconds of maximal static activation of the knee extensors. Horita and Ishiko (1987) also reported a similar decrement of 42% to EMD of the vastus lateralis (117.9 ± 13.4 ms vs. 167.6 ± 28.6 ms) subsequent to 60 seconds of maximal isokinetic knee extensions (180° (3.15) $rad.s^{-1}$), which was also significantly correlated with the accumulation of muscle lactate. Despite the knee flexors offering greatest dynamic protection to the ACL (Rees, 1994), there has been limited scrutiny of the effects of fatigue on this muscle group. Gleeson et al. (1997) showed that 30 continuous reciprocal maximal isokinetic actions of the knee extensors and flexors (3.15 $rad.s^{-1}$) induced a 16% impairment to knee flexor angle specific torque (0.44 rad knee flexion) and a 11.5% increase in EMD of the biceps femoris. Clearly, any temporal impairment of this

type, coupled with concomitant reductions in the force generating capabilities of the active knee stabilisers, may be associated with an acute decrease in the timely capability to restrain dynamic forces of sufficient magnitude to damage knee ligamentous tissue (Mercer et al., 1998). These data may, however, underestimate the potential impairments experienced by the team sports athlete. For example serial episodes of maximal acute exercise are frequently demanded of the sports performer during competitive match-play (Bangsbo, 1994; McInnes et al., 1995). Zhou et al. (1996) observed up to a 70% increase in EMD of the rectus femoris and vastus lateralis subsequent to four bouts of 30 seconds maximal sprint cycling (please see table 2.1). Extrapolation of these values to a schematic depicting the 300 ms ACL injury threshold (please see figure 2.5) shows that, on addition of potential force generating latencies (unpublished data: Gleeson, personal communication), the fatigued athlete has a substantively reduced 'margin for error' to deliver adequate dynamic protection to the knee joint compared to un-fatigued conditions. Under more unfavourable circumstances, for example, the presence of relatively minor intra-articular swelling can inhibit the performance capabilities of joint musculature (Bolgla and Keskula, 2000; Hopkins and Ingersoll, 2000), forces of sufficient magnitude to damage ligament tissue may remain unrestrained.

Figure 2.5. Total latency of dynamic muscle response of fatigued and un-fatigued muscle.



CMCT: central motor conduction time, PMCT: peripheral motor conduction time
EMD: electromechanical delay, $T_{½}$: time to half peak force.
*$T_{½}$ values selected arbitrarily to estimate likely values for the knee flexors of the conditioned and fatigued athlete (Gleeson, personal communication).

Table 2.1. Example investigations of the effects of exercise on EMD (mean ± SD).

Authors (date)	Exercise protocol	Muscle(s)	EMD pre-Exercise	EMD post-Exercise	% change
Horita and Ishiko (1987)	60s maximal isokinetic knee extensions (3.14 $rad.s^{-1}$)	Vastus lateralis	117.9 ± 13.4	167.6 ± 28.6	42.4% ↑
van Dieen et al. (1991)	70% static MVMA 4s work, 2s rest (15 min, or until <60% MVMA)	Erector spinae	136.8 ± 30.9	133.8 ± 20.2	No significant difference
Vos et al. (1991)	150 x 50% MVMA static knee extensions	Vastus lateralis Vastus medialis Rectus femoris	112 ± 11.5 114 ± 11.6 115 ± 12.2	121 ± 18.5 127 ± 19.0 123 ± 21.5	No significant difference
Zhou et al. (1996)	4 x 30s maximal sprint cycling	Pre-training: Vastus lateralis Rectus femoris Post training: Vastus lateralis Rectus femoris	 34.2 ± 8.3 38.0 ± 8.1 35.3 ± 6.4 34.6 ± 5.6	 56.5 ± 12.5 62.3 ± 23.5 60.2 ± 2.0 58.9 ± 13.5	 65.2% ↑ 64.0% ↑ 70.5% ↑ 70.2% ↑
Gleeson et al. (1997)	30 x maximal isokinetic reciprocal knee extension and flexion (3.14 $rad.s^{-1}$)	Biceps femoris	92.9 ± 11.0	103.6 ± 18.3	11.5% ↑
Gleeson et al. (1998b)	PHISR# (90min) Treadmill run (90min) Shuttle run (90min)	Biceps femoris	101.4 ± 16.4 99.8 ± 13.2 100.3 ± 14.2	114.0 ± 25.4 111.5 ± 25.9 119.1 ± 15.4	12.4% ↑ 11.7% ↑ 18.7% ↑
Mercer et al. (1998)	PIHIET†	Biceps femoris	96.7 ± 9.9	125.9 ± 30.7	30.2% ↑
Yeung et al. (1999)	30 x 5s static MVMA (5s rest)	Vastus medialis	36.9 ± 9.0	51.8 ± 1.6	40.4% ↑
Chan et al. (2001)	6 x 30s static knee extensions (10s rest)	Vastus medialis Vastus lateralis	31.7 ± 10.9 32.1 ± 9.5	40.7 ± 11.8 41.2 ± 11.3	28.4% ↑ 28.3% ↑

#PHISR: Prolonged intermittent high intensity shuttle run (1 cycle: 3 x 20m walking, 1 x 15m sprint, 5m walk, 3 x 20m jogging, 3 x 20m running); 2 x 45 min.
†PIHIET: Prolonged intermittent high intensity exercise task: Single leg pedaling task (1 cycle: 42s low intensity, 2s pause, 3s sprint, 22s low-moderate intensity, 22s moderate intensity, 15s recovery); 2 x 45 min.

In contrast to exercise protocols involving MVMA, sub-maximal interventions appear to induce minimal physiological perturbance to the performance capability of the neuromuscular system (please see table 2.1). Neither Vos et al. (1991) or van

Dieen et al. (1991) observed significant changes to EMD performance of the knee extensors and erector spinae following exercise protocols of 70% and 50% MVMA, respectively. These data may be partly explained by potential increases in speed of action potential propagation caused by exercise-induced elevation of muscle temperature, as reported by Hollge et al. (1997), countering possible impairments to EMD. In addition, sub-maximal efforts, which are likely to involve a reduced contribution from the fast twitch fatigable motor units by comparison to maximal muscle actions (Garnett and Stephens, 1981; McComas, 1996), may have minimal effects on temporal neuromuscular capabilities. While it is acknowledged that high threshold motor units can be recruited during rapid sub-maximal activation of muscle (McComas, 1996), the muscle activation and relaxation characteristics in the aforementioned studies (van Dieen et al., 1991; Vos et al., 1991) were not explosive, but externally regulated (e.g. 'ramp and hold'). Consequently, it is possible that these protocols may have induced minimal fatigue (absolute peak force data following exercise was not reported).

Acute sub-maximal exercise interventions seem to be associated with minimal physiologic perturbance to the neuromuscular system, however, this type of exercise which is prolonged and interspersed with repeated episodes of high intensity activity as performed by team sports athletes (McInnes et al., 1995) may predispose performers to cumulative deficits to neuromuscular performance capabilities. To date, only two studies have investigated the effects of prolonged high intensity exercise, simulating the physiologic and metabolic demands of team games (soccer), on temporal neuromuscular performance. Both investigations reported cumulative impairment to EMD performance of the biceps femoris

(Gleeson et al., 1998b; Mercer et al., 1998). Mercer et al. (1998) observed impairment to EMD performance subsequent to a prolonged (approximately 90 minutes) intermittent high intensity single leg pedalling task (96.7 ± 9.9, 110.0 ± 11.1 ms, 125.9 ± 30.7 ms, assessments at pre-, mid and post-exercise, respectively) whereas Gleeson et al. (1998b) employed a shuttle run (approximately 90 minutes) protocol (101.4 ± 16.4 ms, 102.4 ± 13.6 ms, 114.0 ± 25.4 ms) (please see table 2.1 for details). In addition to the total 30% and 12% impairments to EMD performance observed at the end of the cycling and shuttle run exercise, respectively, 16% and 18% reductions in angle specific torque (0.44 rad) of the knee flexors was also observed (Mercer et al., 1998; Gleeson et al., 1998b, respectively). These data may be particularly interesting considered concomitantly with the findings of a recent Football Association audit of injuries of all English professional soccer clubs (competitive seasons 1997/1998 – 1999/1999). The cumulative performance decrements observed by Mercer et al. (1998) and Gleeson et al. (1998b) (from pre- to mid- to post-protocol) appear to 'mirror' the incidence of the greatest numbers of injuries sustained during match-play. Twenty percent and 24%, of all injuries were reported to have occurred towards the end of the first and second periods of play, respectively (Hawkins et al., 2001). Together, these data strongly suggest the link between fatigue and injury.

It is evident that the effects of fatigue on temporal neuromuscular performance can be dramatic and these effects may be related substantively to an acute increased risk of knee ligamentous injury (Gleeson et al., 1998b; Mercer et al., 1998). However, if such activity is unaccustomed, then the associated susceptibility to injury may be prolonged considerably beyond the cessation of exercise. Unaccustomed, high

intensity exercise which involves a large eccentric component whereby the muscle is lengthened while developing force (Latash, 1998), can render the involved musculature susceptible to ultrastructural damage (Armstrong et al., 1983; Jones et al., 1989; Byrne and Eston, 1998). Examples of such exercise within team sports include activities involving deceleration, such as twisting, turning, changing direction and landing. The subsequent exercise-induced muscle damage (EIMD) which is frequently associated with intra-muscular swelling (Armstrong et al., 1983; Foley et al., 1999) and a delayed pain response (delayed onset of muscle soreness) (Jones et al., 1989; Brown et al., 1997) may be experienced by the sports performer who recommences training, match play or even rehabilitation following a prolonged period of inactivity or injury. In addition, the associated decrements to neuromuscular performance capabilities of up to 38% and 69% for rate of force development and peak force, respectively (Strojnik and Komi. 1998; Rinard et al., 2000) can persist for several days (Rinard et al., 2000; Nokasa et al., 2001). While impairments to neuromuscular performance induced by means of acute static or concentric muscle actions may require only three to ten minutes to recover to within pre-exercise values (Gleeson et al., 1997; Zhou, 1996), restoration of performance capabilities following EIMD may require up to seven days (McHugh et al., 2000; Rinard et al., 2000).

Since EIMD is thought to be caused by excessive stress on a small number of active fast twitch muscle fibres during eccentric activation (Linnamo et al., 2000; McHugh et al., 2000; Brockett et al., 2001b; Friden and Lieber, 1992), this type of exercise may also, therfore, selectively impair the maximal capability of the neuromuscular system to maintain joint integrity during mechanical loading. The effects of muscle

damage on the susceptibility to further injury is relatively unknown, however, the findings of the Football Association audit of injuries of all English professional soccer clubs may suggest an increased risk. It was shown that approximately 13% of all injuries sustained over two competitive seasons (1997/1998 – 1999/1999) occurred during pre-season training (Hawkins et al., 2001). Given that physical conditioning can attenuate the symptoms of muscle damage (McHugh et al., 1999), two months of potential 'detraining' during the off-season may render players more susceptible to muscle damage and perhaps to further injury.

The focus of current literature highlights the potential importance of EMD within a conceptual model for knee joint stability (Gleeson et al., 1997; Gleeson et al., 1998a; 1998b; Mercer et al., 1998; Gleeson et al., 2000), however, there has been limited investigation into muscle temporal capabilities following EIMD. To date, the only study that has focussed on the effects of EIMD on EMD showed that sub-maximal stretch-shortening exercise was not associated with impairment to electrically evoked EMD of the knee extensors, despite considerable impairment to volitional peak force (19% decrease) and rate of force development capabilities (38% decrease) (Strojnik and Komi. 2000). A proposed consequence of eccentric EIMD involves muscle length adaptations, where a shift in the length-tension relationship towards longer muscle lengths suggests a shortening of the involved musculature (Morgan and Allen, 1999). Given that the latency of EMD is a function of the interaction of the contractile component with the SEC (Komi, 1979), any shortening of the musculature may effect a greater residual stretch on the SEC at a given joint angle, offsetting potential impairments to EMD caused by possible fast twitch fibre damage. Furthermore, since the aforementioned study utilised

electrical stimulation over the muscle belly to measure EMD performance, activation of the entire muscle, including the damaged fibres, may not have been achieved.

In addition to the EIMD-related impairments to muscle force generating capabilities, losses to neuromuscular control mechanisms, as measured by proprioceptive angle and force replication tasks, has also been demonstrated following eccentric exercise. Saxton et al. (1995) required that participants replicate 35% MVMA in the elbow flexors and two joint angles following 50 maximal eccentric muscle actions, using the control arm as a reference. The results showed that eccentric exercise caused an underestimation of absolute force and joint position. If eccentric exercise is associated with a general suppression of neuromuscular capabilities, including EMD, the timely production of meaningful levels of force may be prevented under conditions of muscle damage, rendering the sports performer at increased risk of ligamentous injury. However, further examination of the data reported by Saxton et al. (1995) may suggest a sophisticated compensatory strategy employed to limit further damage. Calculations revealed that the absolute force produced during the replication tasks following EIMD was 35% of the daily impaired peak force value. Further research investigating temporal neuromuscular responses may help elucidate further possible protective mechanisms. Given that the associated EIMD pain response may follow a quicker time course of recovery compared to neuromuscular performance capabilities (Rinard et al., 2000) and the subsequent possibility of a premature return to competitive match-play, such compensatory mechanisms may be essential to the preservation of knee joint integrity.

While acute exercise has the potential to induce substantive impairments to neuromuscular performance and possible injury avoidance capabilities, if such decrements to performance are a result of predominately concentric or static activities, restoration of neuromuscular performance capabilities may be expected to follow a quick time course of recovery. Unaccustomed exercise involving eccentric activation of muscle, however, may represent a 'worst-case' scenario for the sports performer, since potential decrements to performance capabilities may be prolonged for several days following cessation of activity. These data may also suggest a prolonged susceptibility to ligamentous injury. This may be of particular concern to the sports performer during tournament situations.

2.7 *Summary*

The current review highlights the potential importance of timely corrections to conceivable joint perturbations in order to protect against ligamentous injury. In particular, EMD, may be of principle importance, since this index alone comprises a substantive proportion of neurophysiological latency of response, even under un-fatigued conditions. A wide range of EMD values are reported in the literature, however, it is likely that differential effects of diverse methodologies between studies are a primary cause. Further investigation into the reproducibility and reliability characteristics of this index will elucidate further the associated measurement sensitivity characteristics more fully. The effects of exercise on temporal neuromuscular performance can be profound, although, it is yet to be established whether exercise-related increases in EMD are actually associated with a reduction in the performance capacity of the neuromuscular system as measured

by magnetic stimulation. This method of assessment may be particularly informative in scenarios of exercise-induced muscle damage where selective damage to fast twitch fibres is speculated (McHugh et al., 2000; Brockett et al., 2001b).

Chapter 3
General Methods

3.0 GENERAL METHODS

Protocols for assessment of neuromuscular performance

3.1 *Participants*

Following habituation procedures, each participant completed a standardised warm-up consisting of five minutes cycle ergometry (90 Watts for males, 60 Watts for females) and a further five minutes of static stretching of the involved musculature prior to testing for each study. Participants were instructed to refrain from strenuous physical activity for the 24 hours prior to the test and to maintain constant exercise levels throughout the experimental period, where appropriate. Inter-day assessments of performance were completed as near to the same time of day as possible (± 1 hour). The assessment protocols were approved by the University of Wales, Bangor, Human Performance Ethics Review Committee.

3.2 *Assessment of the knee flexors*

3.2.1 *Participant and dynamometer orientation*

Participants were secured in a prone position on a custom-built dynamometer (modified from Gleeson et al., 1995). The lever-arms of the dynamometer were attached to the legs of the participant via strapping just proximal to the lateral malleolus. The axis of rotation of the dynamometer was aligned midway between the lateral condyle of the tibia and the lateral epicondyle of the femur consistent with the anatomical axis of rotation of the knee joint. The lever-arm length between the ankle cuff and the axis of rotation was standardised for each participant during inter-day trials, where appropriate. To localise the action of the knee flexors, the

trunk was fixed to the apparatus with velcro strapping across the mid thoracic spine and the gluteals, additional immovable restraints were also placed on the posterior of both knees. A knee flexion angle of 25 degrees (0.44 rad), identified during muscle loading, was maintained for both knees throughout testing, recognised as a functionally relevant angle where key ligamentous structures are under greatest mechanical strain (Beynnon and Johnson, 1996) (please see figure 3.1).

3.2.2 *Volitional muscle activation*

After a verbal warning, an auditory signal was delivered to the participant randomly within 1-4 seconds. On receipt of the signal, the participant attempted to flex the knee joint as rapidly and forcefully as possible against the immovable restraint offered by the apparatus. After a period of maximal voluntary muscle activation (MVMA) of at least 2 seconds another auditory signal was delivered to the participant cueing conscious withdrawal of muscle activation and associated neuromuscular relaxation as quickly as possible. Intra-trial MVMA replicates were each separated by at least 10 seconds to enable neuromuscular recovery (Moore and Kukulka, 1991).

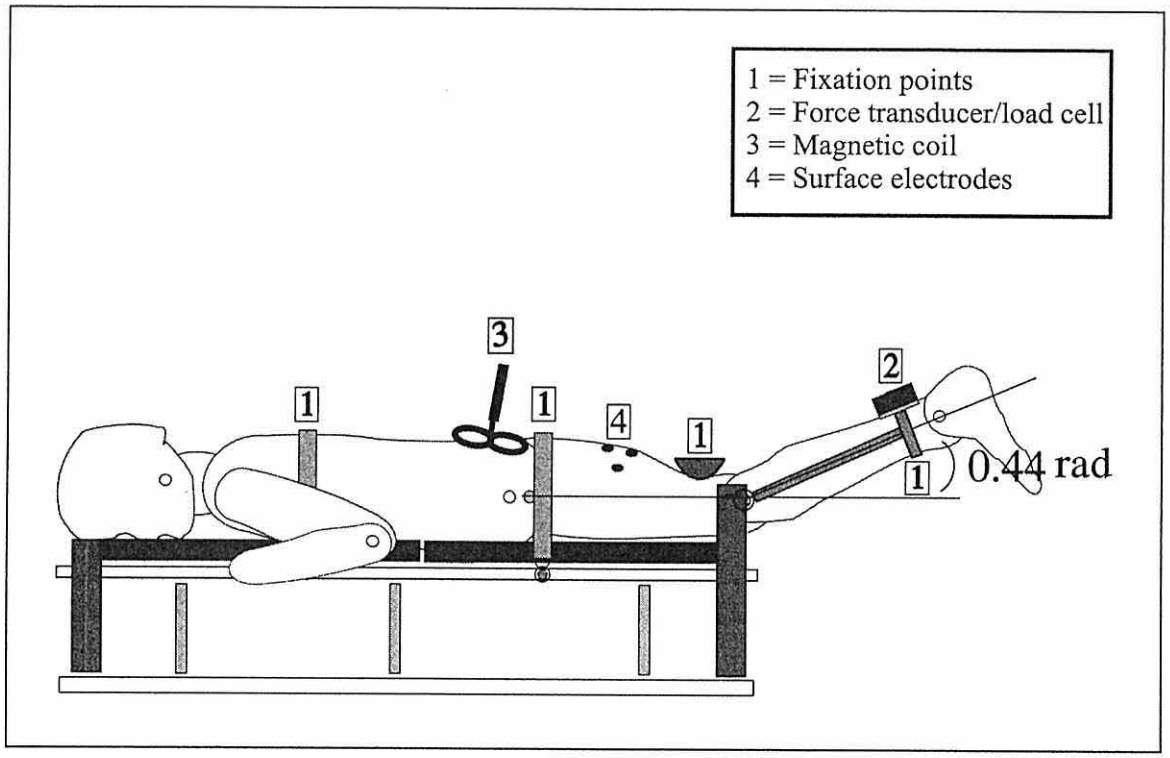
3.2.3 *Magnetically evoked muscle activation*

The sciatic nerve root was stimulated by means of a double wound coil (120 mm), that was powered by a Magstim 200 stimulator (Magstim Co. Ltd., Whitland, Dyfed, Wales) to determine the static neuromuscular performance capacity of the knee flexors associated with a magnetically evoked twitch. The centre of the coil

was placed initially in a position 20 mm to 40 mm lateral to the fifth lumbar vertebra on the involved side. The location of the optimum site for stimulation was denoted by the compound muscle action potential (CMAP) that had the largest amplitude. This was identified by a subsequent procedure in which small positional changes of the coil were made in response to the effects of a series of single stimulations. This optimised coil position was maintained manually throughout the remainder of the test. The optimisation procedure for the site of stimulation was repeated on each test occasion in which participants were required to be re-secured to the dynamometer. A sequence of seven stimulations of increasing intensity was performed to identify supramaximal stimulation. This was defined by a plateauing of the CMAP amplitude. The sequence comprised stimuli at 40%, 50%, 60%, 70%, 80%, 90% and 100%, of the Magstim 200's maximal capacity output and each was separated by at least 10 seconds to enable neuromuscular recovery (Moore and Kukulka, 1991). The beginning of the plateau of the CMAP was defined as the intensity at which no more than a 5% increase in CMAP peak amplitude was observed despite a 10% increase in the intensity of stimulation. This was verified by contemporaneous visual inspection of the data. Supramaximal stimulation was confirmed in 34 of 43 participants. On occasions where supramaximal stimulation was not achieved by this criteria, supplementary tests of peak twitch force (P_TF_E) and electromechanical delay (EMD_E) showed no incremental changes (>5%) from 80% of the Magstim 200's maximal capacity output, indicative of a 'peak' response. Subsequent assessments of magnetically evoked neuromuscular performance in all participants were conducted at a stimulation intensity that was associated with either supramaximal amplitudes of CMAP or peak amplitudes of CMAP limited only by the technological performance of the stimulation system.

Sequential stimulations throughout the experimental period were separated by at least 10 seconds (Moore and Kukulka, 1991).

Figure 3.1. Participant and dynamometer orientation for assessment of the knee flexors.



3.3 *Assessment of the knee extensors*

3.3.1 *Participant and dynamometer orientation*

Participants were secured in a supine position on a custom-built dynamometer (modified from Gleeson et al., 1995). The lever-arms of the dynamometer were attached to the legs of the participant via strapping just proximal to the lateral malleolus. The axis of rotation of the dynamometer was aligned midway between the lateral condyle of the tibia and the lateral epicondyle of the femur consistent with the anatomical axis of rotation of the knee joint. The distance between the ankle cuff and the axis of rotation was standardised for each participant during

inter-day trials, where appropriate. To localise the action of the knee extensors, the trunk was fixed to the apparatus with velcro strapping across the mid thoracic spine and the pelvis, additional immovable restraints were applied to the thigh proximal to the knee. A knee flexion angle of 25 degrees (0.44 rad), identified during muscle loading, was maintained for both knees throughout testing, recognised as a functionally relevant angle where key ligamentous structures are under greatest mechanical strain (Beynnon and Johnson, 1996) (please see figure 3.2).

3.3.2 *Volitional muscle activation*

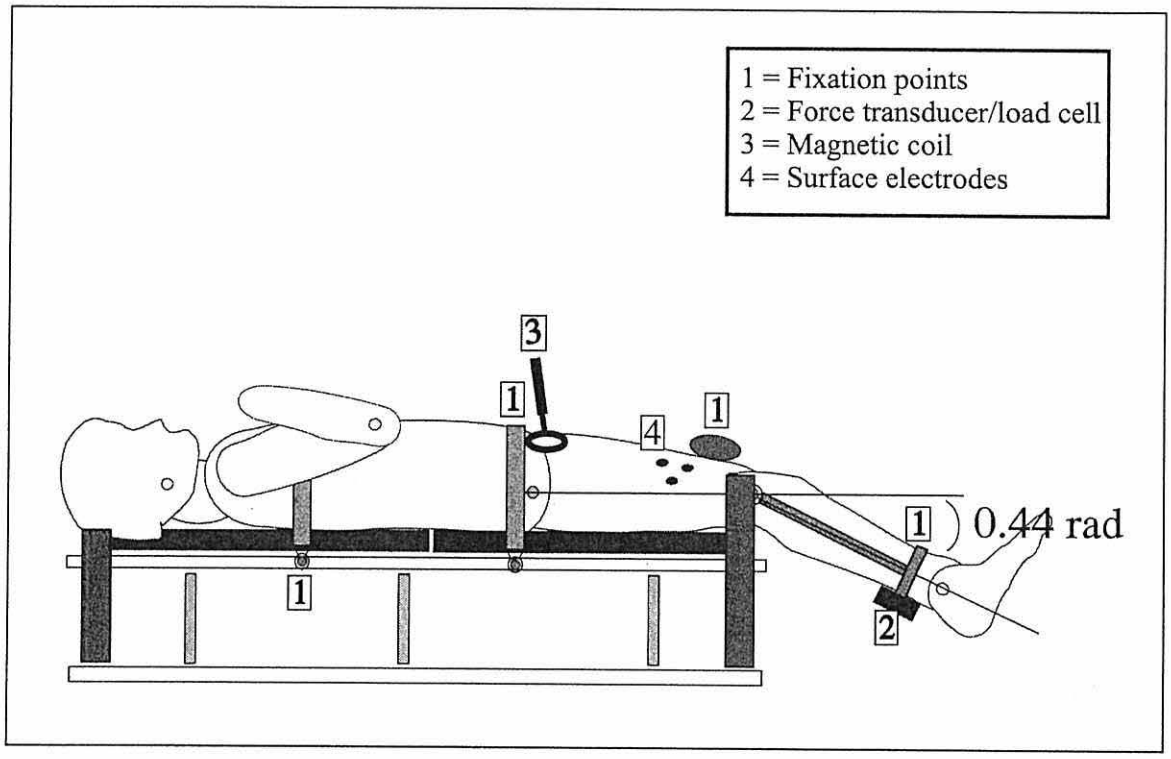
After a verbal warning, an auditory signal was delivered to the participant randomly within 1-4 seconds. On receipt of the signal, the participant attempted to extend the knee joint as rapidly and forcefully as possible against the immovable restraint offered by the apparatus. After a period of maximal voluntary muscle activation MVMA of at least 2 seconds another auditory signal was delivered to the participant cueing conscious withdrawal of muscle activation and associated neuromuscular relaxation as quickly as possible. Intra-trial MVMA replicates were each separated by at least 10 seconds to enable neuromuscular recovery (Moore and Kukulka, 1991).

3.3.3 *Magnetically evoked muscle activation*

The femoral nerve was stimulated by means of double wound coil (120 mm), that was powered by a Magstim 200 stimulator (Magstim Co. Ltd., Whitland, Dyfed, Wales) to determine the static neuromuscular performance capacity of the knee

flexors associated with a magnetically evoked twitch. The centre of the coil was initially placed in the femoral triangle of the involved side. The location of the optimum site for stimulation was denoted by the compound muscle action potential (CMAP) that had the largest amplitude. This was identified by a subsequent procedure in which small positional changes of the coil were made in response to the effects of a series of single stimulations. This optimised coil position was maintained manually throughout the remainder of the test. The optimisation procedure for the site of stimulation was repeated on each test occasion in which participants were required to be re-secured to the dynamometer. A sequence of seven stimulations of increasing intensity was performed to identify supramaximal stimulation. This was defined by a plateauing of the CMAP amplitude. The sequence comprised stimuli at 40%, 50%, 60%, 70%, 80%, 90% and 100%, of the Magstim 200's maximal capacity output and each was separated by at least 10 seconds to enable neuromuscular recovery (Moore and Kukulka, 1991). The beginning of the plateau of the CMAP was defined as the intensity at which no more than a 5% increase in CMAP peak amplitude was observed despite a 10% increases in the intensity of stimulation. This was verified by contemporaneous visual inspection of the data. Supramaximal stimulation was confirmed in 7 of 7 participants. Subsequent assessments of magnetically evoked neuromuscular performance in all participants were conducted at a stimulation intensity that was associated with supramaximal amplitudes of CMAP. Sequential stimulations throughout the experimental period were separated by at least 10 seconds (Moore and Kukulka, 1991).

Figure 3.2. Participant and dynamometer orientation for assessment of knee extensors.



3.4 *Equipment and calibration*

3.4.1 *Force dynamometer*

The load cells of the dynamometer (RDP Electronics Ltd., Wolverhampton, U.K.: range 1000N) were interfaced to a voltage signal recording system that provided analogue to digital conversion of muscular force (Cambridge Electronic Design Ltd., U.K.: 1902 medically isolated programmable amplification/filter [zero amplification] 1401 plus laboratory I/O interface [12-bit ADC sampling at 2 kHz]).

3.4.2 *Test apparatus calibration*

Prior to and repeatedly during testing the technical error performance of the measuring instrument was subjected to a limited validity assessment using inert

gravitational loading. Experimentally recorded force transducer responses were compared to those expected during the application of standard known masses through a biologically valid range (0 N – 600 N). Recorded forces demonstrated an overall mean technical error (± standard error of the estimate) 0.31 N ± 0.03 N across a total of 15 calibrations.

3.5 *Indices of volitional neuromuscular performance*

Estimates of volitional neuromuscular performance were calculated for each MVMA in accordance with the definitions outlined below. The mean values across intra-trial replicates were calculated for every index and were used to describe performance. Participants were not given feedback of results until after the completion of the prescribed number of trials

3.5.1 *Indices of volitional muscle force*

Volitional static peak force (PF_V) was described as the mean response associated with the prescribed number of intra-trial replicates where the highest force was recorded. The average rate of force increase associated with the force-time response between 25 percent and 75 percent of peak force was calculated for each MVMA and the index of rate of force development (RFD_V) was described as the mean response across the intra-trial replicates. The time interval in milliseconds between the observed development of muscle force and the point in time where 50 percent of peak force was achieved was calculated for each MVMA and the index of time to half peak force ($T_{½V}$) was described as the mean response across the intra-trial replicates. Onset of muscle force was defined as the first point in time at

which the force record exceeded consistently the 95% confidence limits associated with the electrical noise amplitude of the load cells. The technical error associated with force transducers was estimated using ordinary statistical procedures and described by the 95% confidence limits that are likely to include the true force score. Estimates based on the background electrical noise of both the right and left force transducers and calculated using 2-second time periods from within a sample of 40 randomly selected data files, showed that the mean (± SD) 95% confidence limit was 1.065 (± 0.097) N. Rate of force development and time to half peak force were selected as indices of the rate at which muscle activation can be initiated and the rapidity with which physiologically meaningful levels of force can be developed.

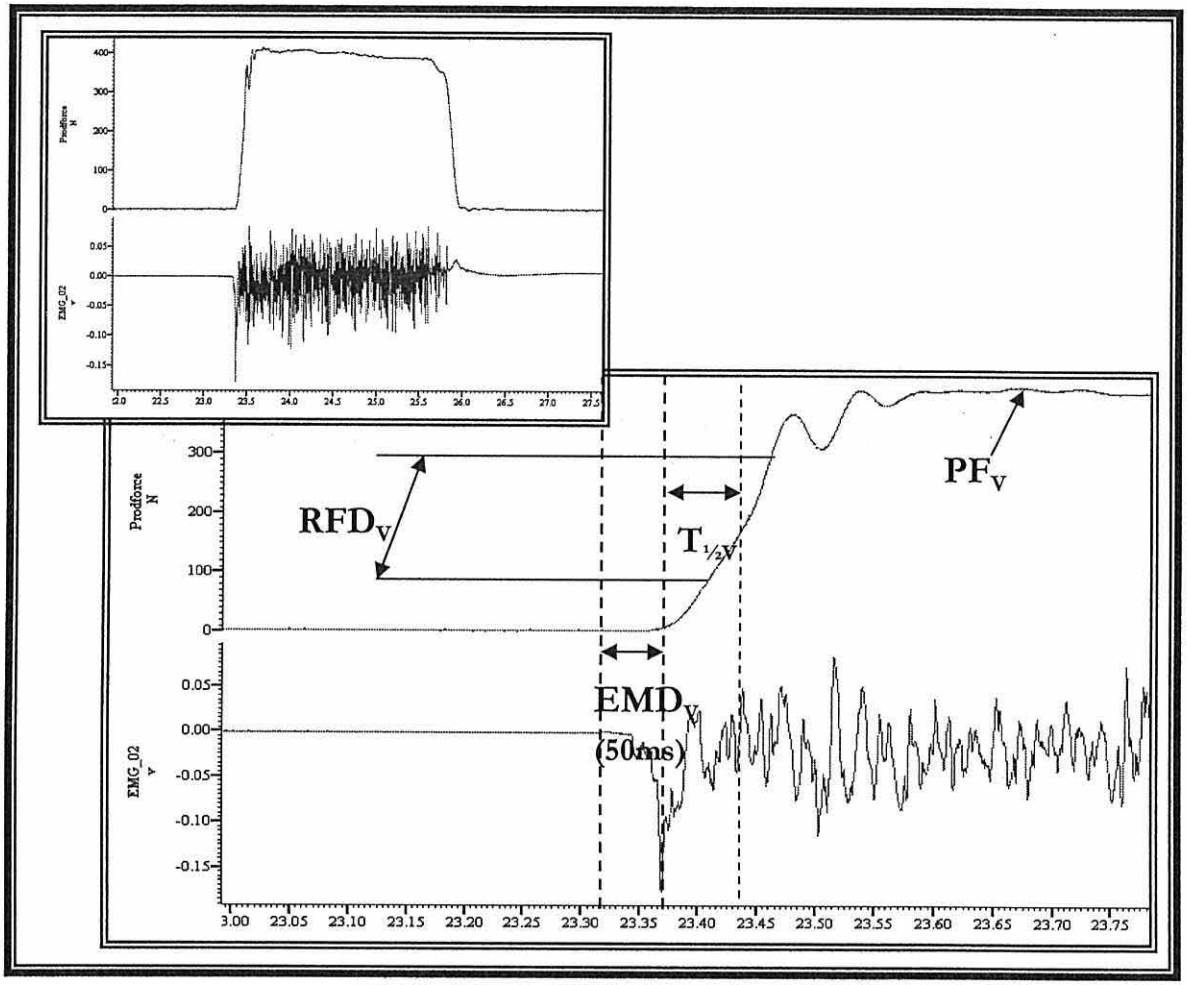
3.5.2 *Indices of electromechanical delay*

The time delay between the onset of electrical activity to the onset of force was calculated for each MVMA. The mean response associated with the prescribed number of intra-trial replicates was used to define volitional EMD (EMD_V). Onset of electrical activity was visually identified as the first deviation of the isoelectric line away from the background noise observed during muscle relaxation. Onset of muscle force was defined as the first point in time at which the force record exceeded consistently the 95% confidence limits associated with the electrical noise amplitude of the load cells. Electromyographic activity (EMG) was recorded with bipolar surface electrodes (self-adhesive, silver-silver chloride, 10mm diameter) which were applied either: longitudinally over the belly of m. biceps femoris, on the line between the ischial tuberosity and the lateral epicondyle of the femur for knee

flexor assessment, or: longitudinally over the distal aspect of the muscle for the m. vastus lateralis on occasions of knee extensor assessment. The m. biceps femoris was selected as a contributor to restraint of anterior tibio-femoral displacement and lateral rotation of the femur relative to the tibia, both implicated in ACL injury (Fu et al., 1993). The m. vastus lateralis was selected to facilitate the distal placement of electrodes away from the magnetic stimulation site in order to minimise the interference of the 'shock' artefact with the muscle electrical response. This limitation has been noted in conjunction with the magnetically evoked assessment of the knee extensors in a previous investigation where electrodes were situated over the belly of m. rectus femoris (Polkey et al., 1996). Electrode placement was standardised across days, where appropriate, by means of mapping (using acetate paper) and measuring the position relative to anatomical landmarks and angiomas. The raw EMG signals underwent amplification (gain = 1000) and analogue to digital conversion at 2 kHz, ensuring a significant margin of safety between the highest frequency expected in the sample and the Nyquist frequency (Gleeson, 2001). The inter-electrode distance was 3 cm and a reference electrode was placed 3cm lateral and equidistant from the recording electrodes. Standard skin preparation techniques yielded inter-electrode impedance of less than 5 kΩ.

Software (Spike 2, version 2.01, Cambridge Electronic Design Ltd., U.K.) was used to interrogate the force and electromyographic data records of volitional muscle activation.

Figure 3.3. Example data showing; upper trace: example data of force and EMG associated with one MVMA; lower trace: magnification of muscle activation to show representative calculation of indices of volitional neuromuscular performance.



3.6 *Indices of magnetically evoked neuromuscular performance*

Estimates of magnetically evoked neuromuscular performance were calculated for each stimulation at the highest intensity.

3.6.1 *Indices of magnetically evoked muscle force*

Static peak twitch force (P_TF_E) was described as the mean response associated with the prescribed number of intra-trial replicates where the highest force was recorded. The average rate of force increase associated with the force-time response between

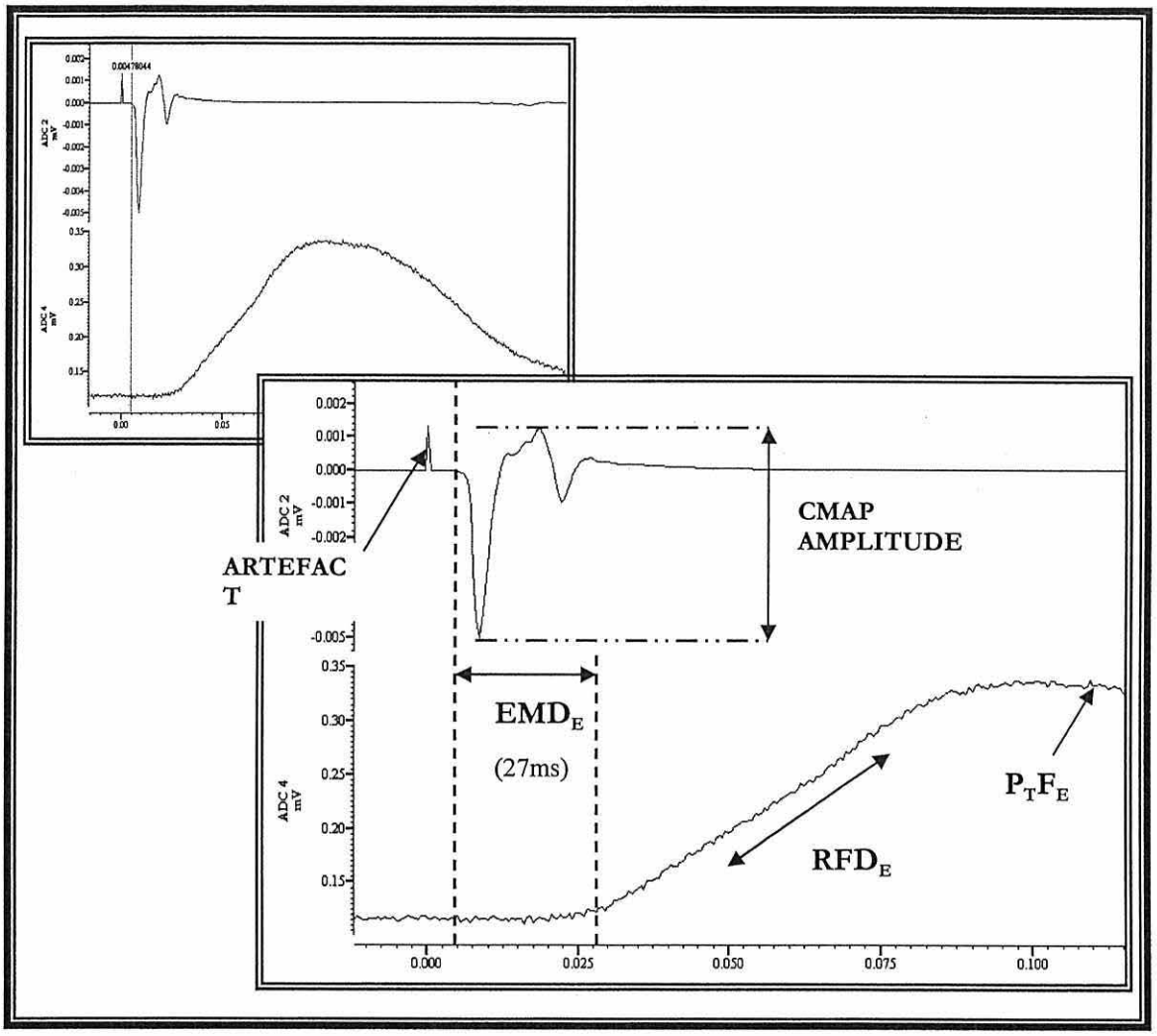
25 and 75 percent of peak twitch force was calculated for each replicate and the mean response across the intra-trial replicates was used to describe the index rate of force development (RFD_E). The time interval in milliseconds between the observed development of peak twitch force and the point in time where 50 percent of peak twitch force was achieved was calculated for each replicate and the mean response across the intra-trial replicates was used to describe the index of time to half peak force ($T_{½E}$). Onset of muscle force was defined as the first point in time at which the force record exceeded consistently the 95% confidence limits associated with the electrical noise amplitude of the load cells (± 0.94 (± 0.13) N; [mean ± SD]).

3.6.2 *Indices of magnetically evoked electromechanical delay*

The time delay between the onset of electrical activity to the onset of force was calculated for each replicate and the mean response associated with the prescribed number of intra-trial replicates was used to describe the index electromechanical delay (EMD_E). Onset of electrical activity was visually identified as the first deviation of the isoelectric line away from the background noise observed during muscle relaxation. Onset of muscle force was defined as the first point in time at which the force record exceeded consistently the 95% confidence limits associated with the electrical noise amplitude of the load cells.

Software (Signal, version 1.81, Cambridge Electronic Design Ltd., U.K.) was used to interrogate the force and electromyographic data records of magnetically evoked muscle activation.

Figure 3.4. Example data showing; upper trace: example data of force and EMG associated with a single magnetic stimulus; lower trace: magnification of muscle activation to show representative calculation of indices of magnetically evoked neuromuscular performance.



Kinanthropometry:

Chapter 4

SINGLE MEASUREMENT RELIABILITY AND REPRODUCIBILITY OF VOLITIONAL AND MAGNETICALLY EVOKED INDICES OF NEUROMUSCULAR PERFORMANCE IN MALES AND FEMALES

Aspects of this work have been presented at the British Association of Sport and Exercise Sciences conference, Newport, 2001, and the European College of Sport Sciences, Athens 2002

Minshull, C., Gleeson, N.P., Rees, D. and Walters, M. (2001). Reproducibility of voluntary and magnetically evoked indices of neuromuscular performance in men and women. British Association of Sport and Exercise Sciences conference, Newport, 2001.
Minshull, C., Gleeson, N.P., Walters, M. and Rees, D. (2002). Reproducibility of voluntary and magnetically evoked indices of electromechanical delay. European College of Sport Sciences, Athens 2002.

4.1 Abstract

The aim of this study was to document the reproducibility and single measurement reliability of indices of neuromuscular performance elicited during electrophysiological activation of the motor pathways by non-invasive magnetic stimulation. Such indices are becoming more popular as adjuncts to established indices of neuromuscular performance associated with maximum volitional efforts in sports medicine applications in the diagnosis of thigh muscle weakness and dysfunction (Gleeson et al., 2000). Seven men (age: 29.6 ± 10.4 yrs; height 1.78 ± 0.04 m; mass 77.0 ± 7.7 kg (mean ± SD) and nine women (age 25.2 ± 4.2 yrs; height 1.69 ± 0.08 m; mass 62.8 ± 8.1 kg) each completed three assessment sessions on three days. At least two minutes and three days separated intra-day and inter-session assessments, respectively. Within each session, assessments of volitional and magnetically evoked indices of neuromuscular performance were obtained from the knee flexors of the preferred leg. Intra-session and inter-day measurement reproducibility was estimated by coefficient of variation (V%, adjusted for small-sample bias) and single measurement reliability by means of intra-class correlations (R_I) and standard error of a single measurement (SEM%) (95% confidence limits). Analysis of V% scores showed equivocal measurement variability between sexes for all indices of volitional and magnetically evoked neuromuscular performance during intra-session and inter-day assessments ($p < 0.05$). As such, judgements about the design of a measurement protocol and its associated level of measurement precision can be made on the same basis for both sexes. Apart from indices of peak force (where magnetically evoked peak twitch force showed greater measurement error), comparisons of the modes of muscle activation show that magnetically

evoked indices of neuromuscular performance offer statistically equivocal levels of measurement reproducibility compared to traditional volitional methods during intra-session and inter-day assessments in the current sample of recreational athletes. Accordingly, the current data lends support to this method of assessment of neuromuscular performance. The results of intra-session and inter-day comparisons show that measurement precision during inter-day comparisons was significantly inferior by contrast to intra-session assessments. Indices of volitional peak force (PF_V) electromechanical delay (EMD_V) and magnetically evoked electromechanical delay (EMD_E) offer the greatest practical utility for the assessment of neuromuscular performance. However, reproducibility and reliability results indicate that single trial protocols do not offer sufficient measurement precision during intra-session (apart from the index PF_V) or inter-day assessments, either for intra-subject or intra-group performance comparisons.

4.2 Introduction

Contemporary empirical research is typically concerned with investigating differences in performance capabilities associated with within-session and between-day assessments (e.g. Beard et al., 1993; Caraffa et al., 1996; Wedderkopp et al., 1999). The goal of such evaluations may be to ascertain the presence of contralateral differences and monitor the effects of prolonged interventions, respectively. Case-study investigations, however, demand stringent criteria for precision of measurement in order to make meaningful performance comparisons (Mercer and Gleeson, 2002). Since the performance levels of the elite strength athlete may be expected to vary by only ±5% over the competitive season (Gleeson and Mercer, 1992), the selected indices of performance should be associated with reliability characteristics that ensure sensitivity to small changes in performance capability (Gleeson & Mercer, 1992). The capability to discriminate performance differences within a group can be equally as important as detecting intra-individual differences. During intra-group comparisons, the reliability characteristics of any given index of performance that ensures sensitivity to small changes in performance capacity will facilitate the separation of the performance capabilities of particular individuals from within the group (Gleeson and Mercer, 1996). Quantified by standard error of the measurement (SEM) in conjunction with the intra-class correlation (R_I) (Thomas and Nelson, 1996)), superior measurement reliability may be imperative within clinical settings to enable the effective targeting of limited resources to the individual patients within a group who require further treatment. The purpose of any index of neuromuscular function is to provide a reliable estimate of performance (Gleeson et al., 2002). Indices of neuromuscular

performance, such as peak force, the rate at which muscle force can be initiated (electromechanical delay) and the rapidity with which meaningful levels of force can be mustered (rate of force development; time to half peak force), can provide a profile of an individual's performance capabilities. Such indices of performance may also provide markers of the dynamic protective capabilities available during mechanical loading of a particular joint system (Johansson, 1991; Rees, 1994; Gleeson et al., 1997; Gleeson et al., 1998a; 1998b; Mercer et al., 1998; Gleeson et al., 2000). Methodologically diverse investigations have examined the reproducibility of peak force of the thigh musculature, and have reported intra-day coefficients of variation (V%) of 4.1% (Viitasalo et al., 1980) and inter-day V% scores of 6.6% (Gleeson et al., 2002) for the knee extensors and flexors, respectively. Other indices of neuromuscular performance, such as electromechanical delay (EMD), have been subject to less scrutiny. There have been a wide range of absolute EMD values reported in the literature even for the same muscle (38.0 ms to 106.0 ms for the rectus femoris (Zhou et al., 1996; Vos et al., 1991), respectively). This has been interpreted by some researchers to represent an inherent variability of this index (Bochdansky et al., 2000). This is despite reports of good intra-day reliability ($r = 0.93$ (Viitasalo et al., 1980)) and reasonable measurement reproducibility (V%: 6.1% (Gleeson et al., 1998); 8.2% (Viitasalo et al., 1980)) for this index of performance and the likelihood that diverse methodologies may have differentially influenced the magnitude of the EMD scores. Further investigation is warranted to elucidate the levels of intra-day and inter-day reproducibility and reliability that might be expected of this index in contemporary practice.

Neuromuscular performance capabilities have been traditionally assessed by means of volitional activation of muscle. However, magnetic stimulation has become increasingly used to estimate the performance capacity of the neuromuscular system (e.g. Barker et al., 1987; Evans et al., 1988; Zhu et al., 1992; Chokroverty et al., 1993; Polkey et al., 1996). This technique has become popular due its ability to potentially counteract factors that confound the proper measurement of an individual's true maximal performance capacity during volitional muscular activation consciously or sub-consciously, such as pacing strategies, waning motivation, injury and associated neuromuscular inhibition (Tsuji and Nakamura, 1988; Zhou et al., 1995; Hopkins and Ingersoll, 2000; Gleeson, 2001). Magnetic stimulation of a peripheral motor nerve activates the fast motor units (Maertens de Noordhout, 1991) and thereby provides an estimate of the true performance capacity of the neuromuscular system (King and Chippa, 1989). Indices of neuromuscular performance subsequent to magnetic stimulation can be utilised in the assessment of the integrity of neural pathways and the diagnosis neurological abnormality (Benecke, 1996; Murray, 1991). Only limited information is available in the contemporary scientific literature regarding the reproducibility and reliability characteristics of indices of neuromuscular performance associated with magnetic stimulation. Polkey et al. (1996) reported intra-day and inter-day coefficients of variation of 3.6% and 8.5% respectively, for maximum twitch tension of the knee extensors subsequent to magnetic stimulation of the femoral nerve. While these estimates are similar to the performance variability of peak force following volitional muscle activation (Viitasalo et al., 1980; Gleeson et al., 2002), further investigation is required that considers other potentially important indices of evoked neuromuscular performance such as EMD and time to half peak force.

Despite evidence that shows women may possess inferior absolute neuromuscular performance capabilities by comparison to male counterparts (Bell & Jacobs, 1986; Holloway & Baechle, 1990; Winter & Brookes, 1991; Behm & Sale, 1994), there has been limited investigation only into whether or not the reproducibility and reliability characteristics of neuromuscular performance scores in males and females differ systematically and how this might impact the design of experimental protocols and ultimately measurement efficacy. Previous examinations of indices of isokinetic leg strength have revealed no differences in performance variability between men and women (Gleeson and Mercer, 1992).

The aim of this study is to examine the intra-session and inter-day reproducibility and single measurement reliability of indices of voluntary and magnetically evoked neuromuscular performance in the knee flexors of males and females.

4.3 Methods

4.3.1 *Participants*

Seven men (age: 29.6 (± 10.4) yrs; height 1.78 (± 0.04) m; body mass 77.0 (± 7.7) kg (mean (± SD)) and nine women (age 25.2 (± 4.2) yrs; height 1.69 (± 0.08) m; body mass 62.8 (± 8.1) kg) gave their informed consent to participate in this study. All participants were regularly involved in exercise (at least 3 times per week) and were asymptomatic at the time of assessment. Participants were instructed to refrain from strenuous physical activity for the 24 hours prior to the test. The assessment protocols were approved by the University of Wales, Bangor, Human Performance Ethics Review Committee. The time commitment of each participant within this study (inclusive of studies 5.0 and 6.0) approximated 2.5 hours.

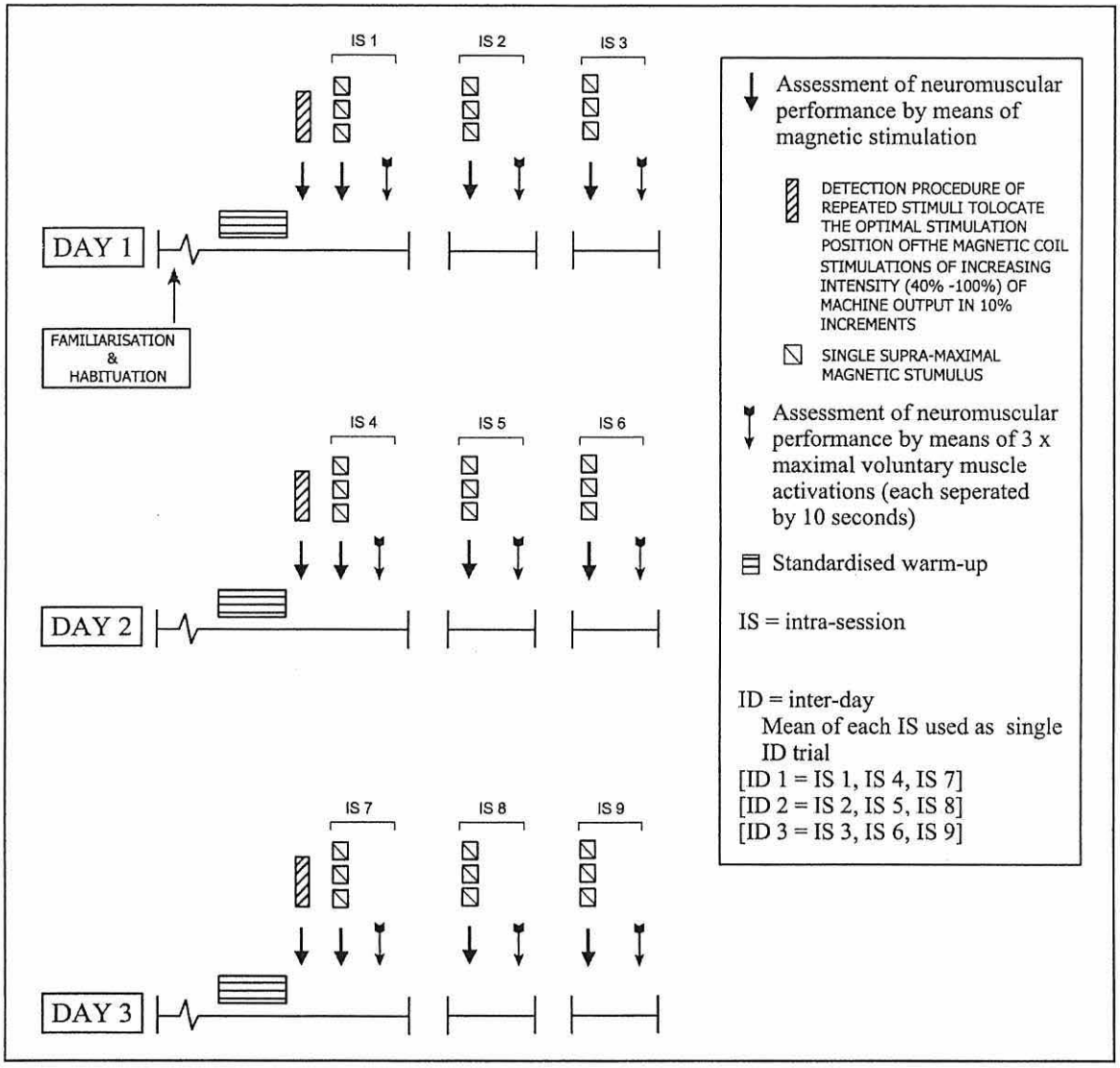
4.3.2 *Experimental procedures*

Following habituation procedures, participants completed a standardised warm-up consisting of five minutes cycle ergometry (90 Watts for males, 60 Watts for females) and a further five minutes of static stretching of the involved musculature prior to testing. Participants were then secured in a prone position on a custom-built dynamometer (modified from Gleeson et al., 1995). The evaluation of volitional and magnetically evoked neuromuscular performance of the knee flexors of the preferred leg were undertaken in accordance with the protocols and experimental conditions outlined in chapter 3.0 (sub-chapter 3.2), please see figure 3.1 for participant and dynamometer orientation (sub-chapter 3.2, p 74).

Assessments of volitional and magnetically evoked neuromuscular performance of the knee flexors of the preferred leg were obtained on three separate days. Inter-

day assessment sessions were separated by at least three days. Within each day, neuromuscular performance was assessed in three separate sessions, each separated by at least 2 minutes. Within each session three estimates of volitional and magnetically evoked performance were obtained. Intra-session trends in absolute performance and measurement reproducibility and reliability were estimated by quantifying the performance variability associated with these three performance scores and was arithmetically averaged over nine possible occasions for intra-session audit (3 sessions x 3 days). Similarly, inter-day trends in absolute performance and estimates of reproducibility and reliability were obtained by quantifying differences and performance variability amongst assessment scores on separate days. A description of the experimental protocol is shown in figure 4.1.

Figure 4.1. Schematic of the protocol for the assessment of intra-session and inter-day measurement reproducibility and reliability for indices of volitional and magnetically evoked neuromuscular performance.



4.3.3 *Indices of volitional neuromuscular performance*

The estimates of volitional neuromuscular performance of the knee flexors of the preferred leg were static peak force (PF_V), electromechanical delay (EMD_V), rate of force development (RFD_V), time to half peak force ($T_{½V}$) and were defined and calculated in accordance with the methods outlined in chapter 3.0 (sub-chapter 3.5).

4.3.4 *Indices of magnetically evoked neuromuscular performance*

The estimates of magnetically evoked neuromuscular performance of the knee flexors of the preferred leg were static peak twitch force (P_TF_E), electromechanical delay (EMD_E), rate of force development (RFD_E) and time to half peak force ($T_{½E}$) and were defined and calculated in accordance with the methods outlined in chapter 3.0 (sub-chapter 3.6).

4.3.5 *Statistical Analyses*

The selected performance indicators were described using ordinary statistical procedures (mean (± SD)). One-way repeated measures analysis of variance (ANOVA) was used to check for systematic learning effects across trials within each testing session (intra-session) and between days (inter-day). Systematic examination of the data revealed that the standard deviation increased in proportion to the mean (heteroscedasticity). As such, coefficient of variation (V%), corrected for small sample bias (Sokal and Rohlf, 1981), was used to assess variability of indices across the three trials for each intra-session and inter-day estimate. Coefficient of variation was calculated according to the expression (SD/mean) x (1+(1/4n) x 100) and expressed as a percentage, where n is the number of trials. The Shapiro-Wilks' test was used to check the normal distribution of V% scores. Single measurement reliability for each index was assessed by computing intra-class coefficients (R_I) and standard error of a single measurement (SEM%) (95% confidence limits), expressed as a percentage of the group mean score according to the formula $((SD \times \sqrt{(1-R_I)})/mean) \times 100$ (multiplied by 1.96 to compute 95% confidence limits and assuming a normal distribution of scores). The Spearman-Brown prediction formula (Winer, 1981) was used to compute the expected

reliability of the mean of multiple measurements for each index. Variability (V%) between groups (male: female) was compared using a two (time: intra-session; inter-day) by two (mode of muscle activation: volitional; evoked) by two (group: males; females) mixed-model ANOVA with repeated measures on the first two factors. Where changes in group mean absolute performance scores across intra-session and inter-day comparisons offered evidence of systematic carry-over effects, these specific assessment occasions were omitted from subsequent analyses of performance reproducibility and single-measurement reliability (Thomas and Nelson, 1996). The assumptions underpinning the use of repeated measures ANOVA were checked and violations corrected by the Greenhouse-Geisser adjustment of the critical F-value, as indicated by $_{GG}$. Statistical significance was accepted at $p < 0.05$.

4.4 Results

The statistical parity across the vast majority of trials suggests that changes in performance can be attributed to technical error and biological variation.

4.4.1 *Results of reproducibility analyses*

Tables 4.1 and 4.2 show V%, R_I and SEM% scores for intra-session and inter-day day measures, respectively, for males and females separately. Repeated measures mixed-model ANOVAs revealed no significant differences in variability (V%, adjusted for small sample bias) between males and females on the tested indices, data was therefore amalgamated to describe measurement precision. Table 4.3 shows the absolute group mean performance scores for males and females (±SD) for intra-session and inter-day measures.

Table 4.1. Intra-session group mean coefficient of variation (V%), intra-class correlation coefficient (R_I) and standard error of the measurement (SEM%) (95% confidence levels, expressed as a percentage of the mean group score) (mean ± SD) for indices of volitional ($_V$) and magnetically evoked ($_E$) neuromuscular performance for males and females.

Index	V%		R_I		SEM%	
	Males	Females	Males	Females	Males	Females
PF_V	2.6 ± 1.3	4.4 ± 1.8	0.97 ± 0.02	0.96 ± 0.03	3.1 ± 1.1	4.6 ± 1.5
P_TF_E	10.9 ± 2.9	12.0 ± 4.3	0.91 ± 0.10	0.82 ± 0.29	10.7 ± 7.3	23.4 ± 22.3
EMD_V	11.8 ± 1.9	9.3 ± 4.1	0.69 ± 0.07	0.88 ± 0.06	12.0 ± 2.9	8.7 ± 1.8
EMD_E	4.6 ± 2.1	4.7 ± 3.0	0.85 ± 0.12	0.74 ± 0.13	6.1 ± 2.6	7.4 ± 1.5
RFD_V	18.9 ± 6.1	19.5 ± 6.9	0.66 ± 0.17	0.81 ± 0.13	14.5 ± 3.9	23.7 ± 8.9
RFD_E	17.6 ± 11.1	22.1 ± 7.8	0.88 ± 0.12	0.90 ± 0.11	17.3 ± 9.9	26.1 ± 24.3
$T_{½V}$	12.5 ± 2.7	15.0 ± 4.5	0.66 ± 0.17	0.47 ± 0.22	14.5 ± 3.9	18.0 ± 7.6
$T_{½E}$	13.4 ± 4.5	13.5 ± 2.6	0.62 ± 0.24	0.66 ± 0.17	15.7 ± 7.9	14.0 ± 5.7

Table 4.2. Inter-day group mean coefficient of variation (V%), intra-class correlation coefficient (R_I) and standard error of the measurement (SEM%) (95% confidence levels, expressed as a percentage of the mean group score) (mean ± SD) for indices of volitional ($_V$) and magnetically evoked ($_E$) neuromuscular performance for males and females.

Index	V%		R_I		SEM%	
	Males	Females	Males	Females	Males	Females
PF_V	8.3 ± 3.9	9.3 ± 3.6	0.79 ± 0.06	0.87 ± 0.04	8.4 ± 2.4	10.3 ± 1.9
P_TF_E	23.4 ± 7.6	23.5 ± 8.0	0.70 ± 0.11	0.76 ± 0.10	28.8 ± 9.0	30.0 ± 8.9
EMD_V	14.8 ± 5.7	14.2 ± 5.7	0.36 ± 0.13	0.76 ± 0.09	16.8 ± 4.9	14.2 ± 2.2
EMD_E	14.5 ± 7.2	12.1 ± 5.6	0.59 ± 0.13	0.24 ± 0.07	15.2 ± 4.0	14.9 ± 3.3
RFD_V	21.2 ± 6.3	28.3 ± 9.8	0.66 ± 0.17	0.59 ± 0.11	14.5 ± 3.9	33.8 ± 5.8
RFD_E	30.2 ± 15.8	29.8 ± 7.4	0.64 ± 0.12	0.78 ± 0.09	33.3 ± 5.2	36.1 ± 9.6
$T_{½V}$	14.9 ± 2.5	17.0 ± 5.9	0.66 ± 0.17	0.36 ± 0.18	14.5 ± 3.9	18.3 ± 5.2
$T_{½E}$	19.2 ± 5.8	19.3 ± 5.1	0.40 ± 0.18	0.29 ± 0.08	19.3 ± 1.9	20.5 ± 5.3

Table 4.3. Absolute group mean intra-session and inter-day scores for indices of volitional ($_V$) and magnetically evoked ($_E$) neuromuscular performance for males and females (mean ± SD).

Index		Intra-session		Inter-day	
		Males	Females	Males	Females
PF_V	(N)	325.1 ± 52.4	195.9 ± 45.6	311.8 ± 52.8	190.8 ± 48.6
P_TF_E	(N)	16.7 ± 6.9	13.6 ± 8.1	14.7 ± 6.8	18.8 ± 6.5
EMD_V	(ms)	47.8 ± 5.9	52.3 ± 13.1	49.5 ± 6.4	50.6 ± 6.3
EMD_E	(ms)	31.7 ± 4.4	26.9 ± 2.5	32.2 ± 4.5	25.3 ± 2.9
RFD_V	($N.s^{-1}$)	2467.1 ± 466	1239.6 ± 530	2364.8 ± 530	1142.9 ± 242
RFD_E	($N.s^{-1}$)	602.5 ± 273	545.1 ± 341	547.7 ± 287	678.1 ± 261
$T_{½V}$	(ms)	81.8 ± 13.4	81.5 ± 12.4	84.7 ± 15.6	117.8 ± 20.5
$T_{½E}$	(ms)	12.2 ± 2.1	11.1 ± 1.6	12.3 ± 2.5	11.4 ± 1.5

Table 4.4 shows group mean coefficient of variation (V%, adjusted for small sample bias), intra-class correlation coefficients (R_I) and standard error of a single measurement (SEM%) (95% confidence levels, expressed as a percentage of the mean group score) for intra-session and inter-day measures.

Table 4.4. Intra-session and inter-day group mean coefficient of variation (V%), intra-class correlation coefficient (R_I) and standard error of the measurement (SEM%) (95% confidence levels, expressed as a percentage of the mean group score) (mean ± SD) for indices of volitional ($_V$) and magnetically evoked ($_E$) neuromuscular performance.

Index	Intra-session			Inter-day		
	V%	R_I	SEM%	V%	R_I	SEM%
PF_V	3.7 ± 1.8	0.98 ± 0.01	4.0 ± 0.9	8.9 ± 3.6	0.93 ± 0.02	8.7 ± 1.2
P_TF_E	11.5 ± 3.7	0.92 ± 0.08	15.2 ± 9.7	23.4 ± 7.6	0.73 ± 0.10	31.6 ± 9.4
EMD_V	10.1 ± 3.4	0.80 ± 0.06	10.8 ± 1.8	14.5 ± 5.5	0.64 ± 0.09	15.9 ± 3.1
EMD_E	8.1 ± 3.9	0.84 ± 0.11	6.9 ± 2.6	13.1 ± 6.3	0.51 ± 0.12	13.5 ± 1.9
RFD_V	19.2 ± 6.3	0.80 ± 0.08	23.8 ± 5.5	25.2 ± 9.0	0.71 ± 0.12	28.8 ± 5.0
RFD_E	20.1 ± 9.3	0.91 ± 0.09	17.8 ± 9.8	30.0 ± 11.4	0.70 ± 0.11	35.9 ± 8.7
$T_{½V}$	13.9 ± 3.9	0.54 ± 0.11	16.8 ± 5.1	15.8 ± 4.7	0.39 ± 0.13	16.2 ± 2.3
$T_{½E}$	13.5 ± 3.9	0.65 ± 0.12	14.9 ± 4.3	19.3 ± 5.2	0.32 ± 0.13	20.3 ± 4.5

Indices of peak force (PF_V/P_TF_E)

A significant two-factor interaction (time by mode of muscle activation) ($F_{[1,14]}$ = 23.4, $p < 0.001$) showed that while greater variability in performance was associated with inter-day compared to intra-session measures, the difference in performance variability between intra-session and inter-day comparisons was larger for magnetically evoked peak twitch force (11.5 (± 3.7)% vs. 23.4 (± 7.6)% (group mean V% (± SD), respectively) compared to volitional peak force (3.7 (± 1.8)% vs. 8.9 (± 3.6)%, respectively) (please see table 4.4).

Indices of electromechanical delay (EMD_V/EMD_E)

A significant main effect for time associated with the repeated measures mixed-model ANOVA ($F_{[1,14]} = 30.4$, $p < 0.001$) revealed greater variability of performance across days (inter-day) by comparison to intra-session. The group mean V% (± SD) scores for EMD_V and EMD_E were 10.1 (± 3.4)% vs. 14.5 (± 5.5)% and 8.1 (± 3.9)% vs. 13.1 (± 6.3)% (intra-session and inter-day, respectively).

Indices of rate of force development (RFD_V/RFD_E)

A significant main effect for time associated with the repeated measures mixed-model ANOVA ($F_{[1,14]} = 28.6$, $p < 0.001$) revealed greater variability of performance across days (inter-day) by comparison to intra-session. The actual group mean V% (± SD) scores for RFD_V and RFD_E were 19.2 (± 6.3)% vs. 25.2 (± 9.0)% and 20.1 (± 9.3)% vs. 30.0 (± 11.4)%, respectively (intra-session and inter-day, respectively).

Indices of time to half peak force ($T_{½V}/T_{½E}$)

A significant two-factor interaction (time by mode of muscle activation) associated with the repeated measures mixed-model ANOVA ($F_{[1,14]} = 23.4$, $p < 0.001$) showed that while inter-day measures were associated with greater variability in performance compared to intra-session measures for both modes of muscle activation, the difference in performance variability between intra-session and inter-day comparisons was larger for $T_{½E}$ (13.5 (± 4.0)% vs. 19.3 (± 5.2)% (group mean V% (± SD), respectively) compared to $T_{½V}$ (13.8 (± 3.9)% vs. 15.9 (± 4.7)%, respectively) (please see table 4.4).

4.4.2 *Results of single measurement reliability analyses*

While the R_I during intra-session measures for most indices (apart from $T_{½V}$ and $T_{½E}$) exceed or approximated a clinically acceptable reliability coefficient threshold of greater than 0.80 (Currier, 1984), some group mean SEM% scores, which compensate for potential over estimation of reliability by taking account of the group heterogeneity, indicates a limited capability to discriminate performance changes based on single-trial assessments associated with intra-group comparisons (Gleeson and Mercer, 1996) (range: 4.0 (± 0.9)% to 23.8 (± 5.5)% (group mean V% ± (SD)), 95% confidence limits) (please see table 4.4). During inter-day measures, however, the R_I for PF_V only exceeded a clinically acceptable reliability coefficient threshold of greater than 0.80 (Currier, 1984), yet, SEM% scores (95% confidence limits) show a limited capability to detect performance differences based on single-trial assessments associated with intra-group comparisons across days (please see table 4.4).

4.5 Discussion

4.5.1 *Precision of measurement associated with intra-session estimates of performance*

The group mean intra-session variability of PF_V and EMD_V of the knee flexors in the present study (V%: 3.7% and 10.1%, respectively), is similar to previously reported coefficients of variation of these indices for the knee extensors (V%: 4.1% and 8.3%, respectively) (Viitasalo et al., 1980). However, the variability of P_TF_E (11.5%) exceeds both the intra-day and inter-day V% scores reported by Polkey et al. (1996) (3.6% and 8.5%, respectively). While the underlying mechanisms for the observed greater variability are currently unclear, a contributing factor may include a possible greater ease in location of the optimal site for stimulation associated with knee extensor activation. Such may affect the portion of the nerve subsequently stimulated and the consistency of magnetically evoked force response. In addition, greater performance variability under evoked compared to volitional conditions may be due to the expression of performance variability in Newtons relative to smaller absolute performance scores. Results show that PF_V is associated with superior measurement reproducibility compared to all other indices of neuromuscular performance (please see table 4.4). The remaining indices demonstrate a compromised capability to discriminate subtle changes in performance during intra-individual comparisons, with coefficients of variation of up to ±20.1% for RFD_E. Calculation of 95% confidence limits, using the central limit theorem (Thomas and Nelson, 1996), reveals a window of error of at best ±7.3% for PF_V and at worst ±39.4% for RFD_E on a single estimate of performance. Very few intra-individual comparisons within a particular test session may be expected to demonstrate performance differences that will exceed a level of single measurement precision of

78.8% (±39.4%). Given the focus of current research highlighting the potential importance of indices other than peak force performance to injury prevention (e.g. Wojtys and Huston, 1996; Gleeson et al., 1998a; Mercer et al., 1998), this lack of precision of measurement may be of particular concern to the clinician trying to ascertain injury risk using the asymptomatic contralateral limb as the comparison. These results challenge the utility of single-trial protocols for the assessment of intra-individual neuromuscular performance differences. Using a criterion based on the central limit theorem that the estimated error of the mean score of multiple trials would be expected to vary inversely with the square root of the number of intra-individual replicates (assuming a normal distribution of errors) (Winer, 1981), the mean score of at best 2 intra-individual replicates for PF_V and at worst > 25 intra-individual replicates for indices and RFD_E and RFD_V would be required to achieve a level of arbitrarily acceptable level of measurement precision of better than ±5% (please see figure 4.2). The equivalent threshold number of trials for EMD_V, EMD_E and P_TF_E are 15, 10 and 20, respectively, with the indices $T_{½V}$ and $T_{½E}$ each requiring the mean of approximately 25 replicates.

Figure 4.2. Error associated with the assessment of RFD_V (open bars) and RFD_E (closed bars) using 1 to 25 intra-session trials: coefficient of variation (V% [95% confidence limits]) and standard error of the measurement (SEM% [95% confidence limits]).

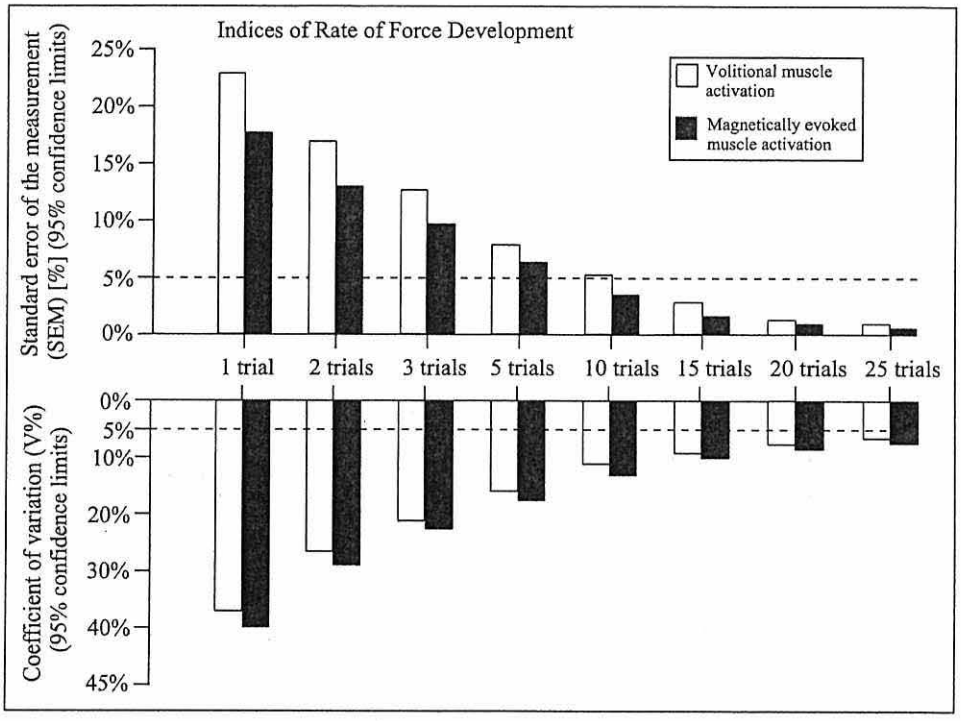
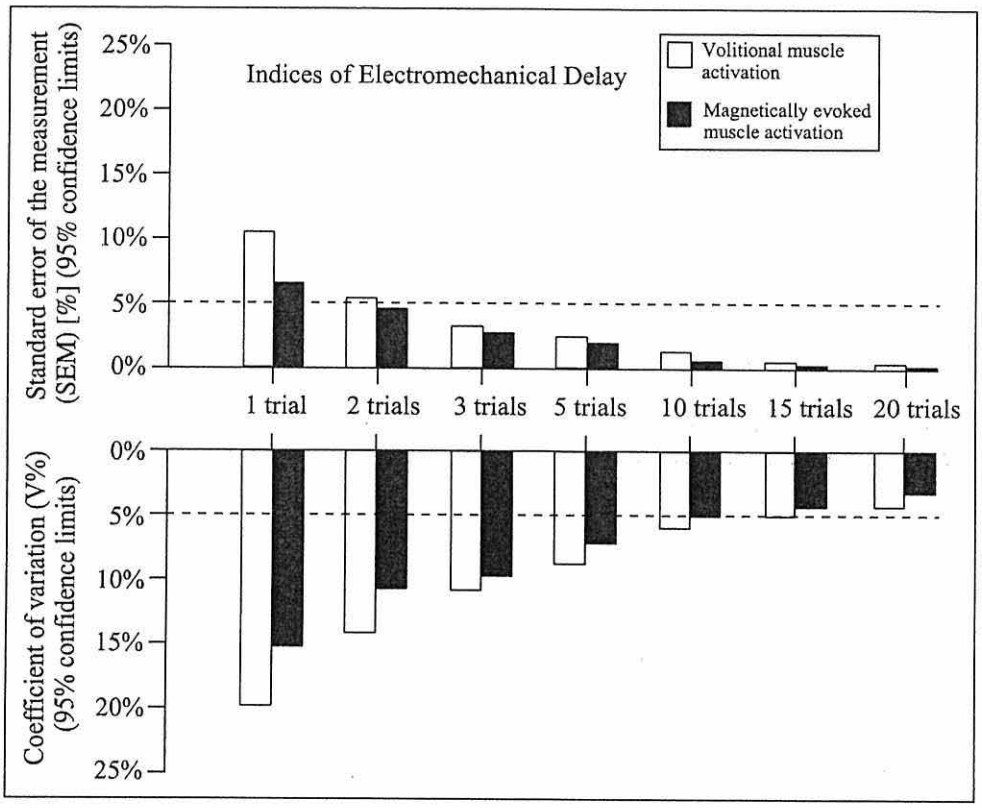


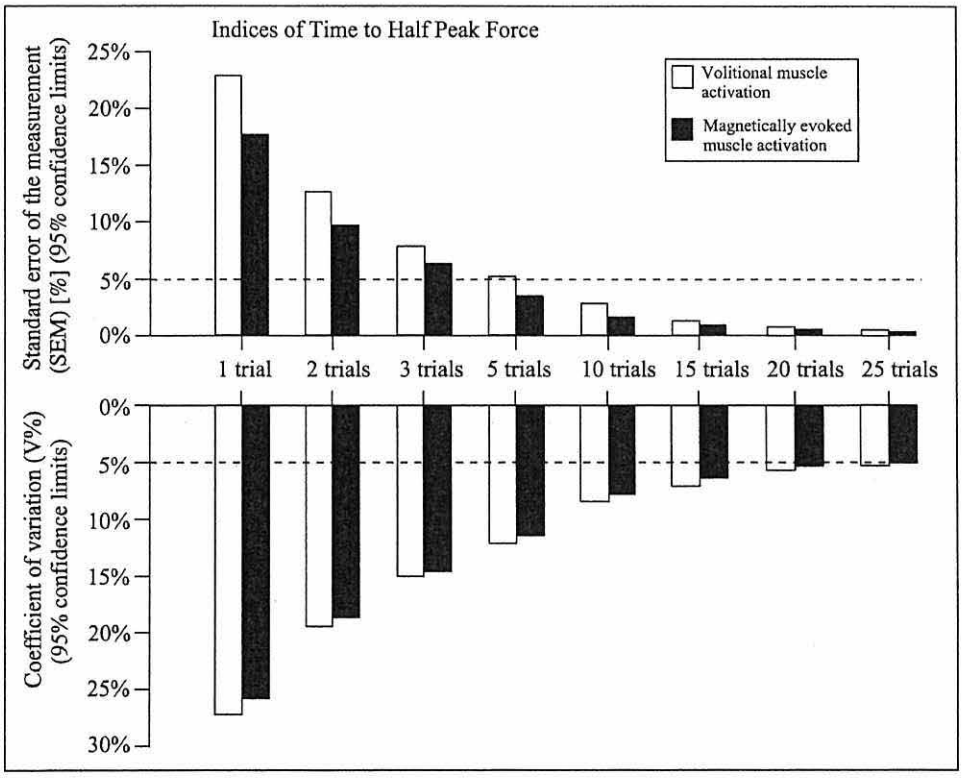
Figure 4.3. Error associated with the assessment of EMD_V (open bars) and EMD_E (closed bars) using 1 to 20 intra-session trials: coefficient of variation (V% [95% confidence limits]) and standard error of the measurement (SEM% [95% confidence limits]).



Despite the R_I scores for most indices (apart from $T_{½V}$ and $T_{½E}$) equalling or exceeding a clinically acceptable reliability coefficient of greater than 0.80 (Currier, 1984), SEM% scores, which takes group performance heterogeneity into account, also indicate a limited capability to discriminate physiological difference based on the achievement of on average, intra-group separation between scores of ±5%. This is the case for all indices apart from PF_V. In scenarios involving inter-group treatment comparisons, where participant numbers can be manipulated to achieve a desired level of experimental power (Lipsey, 1990), single-trial performance assessments may be acceptable. However, identification of individuals within a group possessing intra-individual differences that require further treatment would require a higher level of measurement sensitivity. Such would only currently be

afforded by using a mean score of multiple trials as the basis for estimating performance in order to reduce measurement error (Gleeson et al., 2002). In addition to the index PF_V, the Spearman-Brown prophecy formula (Winer, 1981) used in conjunction with the calculation of SEM% suggests that indices EMD_V and EMD_E offer the greatest practical utility to be able to discriminate properly between individuals within a group, requiring the mean scores of 3 and 2 intra-individual trials, respectively to achieve a level of measurement error of better than ±5%. The equivalent threshold number of trials for $T_{½V}$, $T_{½E}$, RFD_V and RFD_E is >5, 5 10 and >10, respectively. Based on this criterion, indices of time to half peak force should be the preferred estimate of force generating capability over rate of force development.

Figure 4.4. Error associated with the assessment of $T_{½V}$ (open bars) and $T_{½E}$ (closed bars) using 1 to 25 intra-session trials: coefficient of variation (V% [95% confidence limits]) and standard error of the measurement (SEM% [95% confidence limits]).



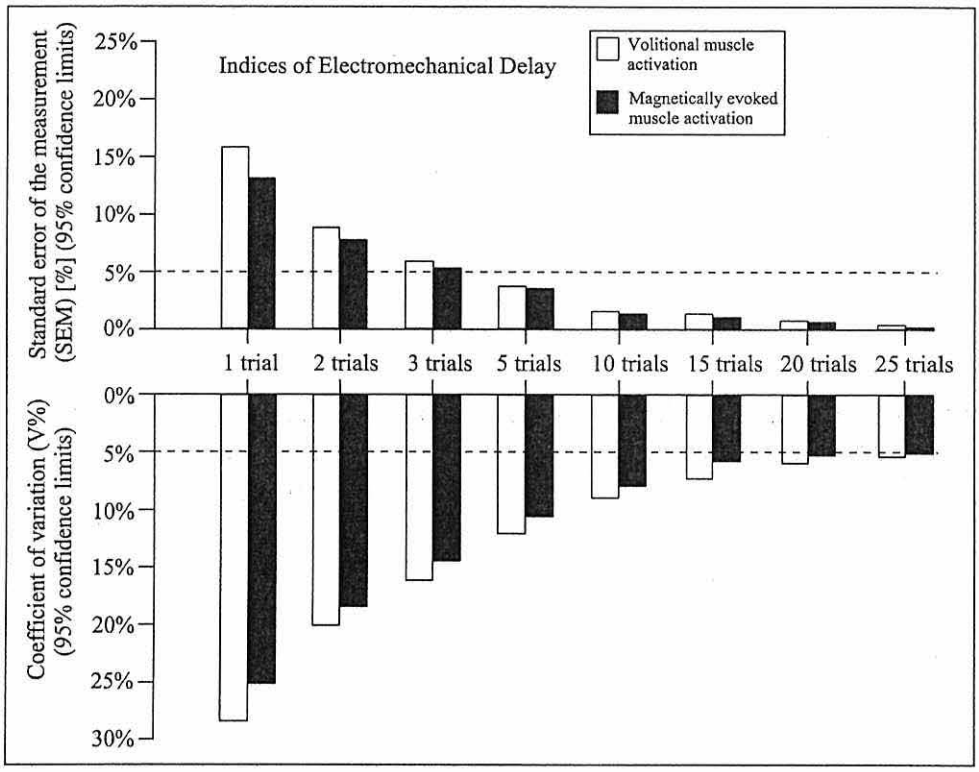
The results of reproducibility and reliability analyses indicate that the index PF_V is associated with the greatest measurement precision during intra-session comparisons. The reproducibility and reliability characteristics of this index make possible the detection of intra-group and intra-individual differences on the basis of 1 and the mean of 2 trials, respectively (95% confidence limits). The indices EMD_V and EMD_E offer reasonable measurement utility, particularly during intra-group comparisons. These results are in contrast to the findings of the limited evaluation conducted by Bochdansky et al. (2000). However, the current findings also show that single-trial protocols are inappropriate for the estimation of within-session differences in performance capabilities, particularly during intra-individual assessments.

4.5.2 *Precision of measurement associated with inter-day estimates of performance*

Analyses revealed that all indices of performance were associated with significantly greater variability (V%) during inter-day compared to intra-session conditions. The variability of PF_V of the knee flexors in the present study (V% = 8.9 ± 3.6%) is greater than previous reports of performance variability of this index (Gleeson et al., 2002). Contributing factors to the high levels of performance variability in the current study might include a lack of familiarity of maximal volitional activation of the knee flexor group, particularly at a 'sub-optimal' knee angle for maximal force production (Brockett et al., 2001). Indeed, investigations have found greater inter-day V% scores for indices of isokinetic leg strength in the knee flexor group comparison to the knee extensors (Gleeson and Mercer, 1992). These factors may

impact the inter-day consistency of static force production. Despite this, table 4.4 reveals that PF_V is associated with superior inter-day measurement reproducibility and reliability compared to all other indices of neuromuscular performance, consistent with the findings of intra-session comparisons. Computation of 95% confidence limits of V% scores reveals that indices PF_V and EMD_E offer the greatest measurement utility for the assessment of intra-individual performance changes between days, requiring the mean score of 15 and 25 trials, respectively to achieve a level of measurement sensitivity of approximately ±5%. The equivalent threshold mean number of trials for all other indices is > 25. Clearly, these results challenge the utility of even the most reproducible of indices to accurately discriminate subtle intra-individual changes in performance during inter-day comparisons.

Figure 4.5. Error associated with the assessment of EMD_V (open bars) and EMD_E (closed bars) using 1 to 25 inter-day trials: coefficient of variation (V% [95% confidence limits]) and standard error of the measurement (SEM% [95% confidence limits]).



The R_I for PF_V only (0.93 ± 0.02) exceeds a clinically acceptable reliability coefficient of greater than 0.80 (Currier, 1984), yet, the group mean SEM% scores indicate a limited capability to detect performance differences associated with intra-group comparisons based on single-trial assessments (8.7 ± 1.2%). However, the Spearman-Brown prophecy formula (Winer, 1981) suggests that the mean of 2 trials only are required to achieve a level of measurement precision better than ±5%. Using this criterion, the indices EMD_V, EMD_E also offer reasonable measurement utility, both requiring the mean of 5 trials to achieve a similar level of measurement error (please see figure 4.5). Despite $T_{½V}$ and $T_{½E}$ offer superior measurement inter-day reliability compared to RFD_V and RFD_E, consistent with the intra-session results, (please see table 4.4), the mean score of 10, (compared to 15 for RFD_V and

RFD_E) intra-subject replicates are still required to obtain an equivalent level of measurement sensitivity. The threshold numbers of trials for P_TF_E is > 25.

These results, while confirming that single-trial protocols are unacceptable for the proper interpretation of neuromuscular performance between days, show the potential for better inter-day measurement utility during intra-group by comparison to intra-individual assessments. Based on the number of trials required to achieve an acceptable level of measurement precision, accurate discrimination of minor intra-individual performance changes may not be possible between days. Indeed, within an elite performance sub-sample, or a patient population approaching optimally rehabilitated status, it may be wholly appropriate to demand a higher level of measurement precision (< ±5%) to confidently discriminate subtle, but potentially clinically important, changes in neuromuscular performance. Such would greatly impact the efficacy of the chosen measurement protocol. Furthermore, since the V% scores relate to the average variability, the performance heterogeneity of some individuals is not fully represented. Accordingly, these results may raise concern regarding the capability to monitor accurately individual neuromuscular performance capabilities in cases where gross differences are not expected.

4.5.3 *Summary*

These data show equivalent measurement variability between sexes for indices of volitional and magnetically evoked neuromuscular performance during intra-session and inter-day assessments. Accordingly, judgements about the design of a

measurement protocol and its associated level of measurement precision can be made on the same basis for both sexes. Apart from indices of peak force, comparisons of the modes of muscle activation show that magnetically evoked indices of neuromuscular performance offer statistically equivalent levels of measurement reproducibility compared to traditional volitional methods during intra-session and inter-day assessments in the current sample of recreational athletes. As such, the current data lends support to this method of assessment of neuromuscular performance. Estimation of muscle performance capacities by means of magnetic stimulation can have advantages over traditional methods, particularly on occasions where an individual's maximal performance capabilities are inhibited by pain and/or intra-articular effusion (Hopkins and Ingersoll, 2000; Gleeson, 2001). However, the exclusive use of magnetic stimulation must be carefully considered by the sports medicine practitioner. While relatively large, detectable contrasts in performance may be associated with such a scenario, the Spearman-Brown prophecy formula (Winer, 1981) suggests that at least 15 and 25 trials are required to discriminate between intra-group P_TF_E scores with a more stringent level of measurement error ($< \pm 5\%$) during intra-session and intra-day assessments, respectively. The index PF_V however, requires only 1 and the mean of 2 trials, respectively to make equivalent comparisons. Despite the relative ease by which multiple trials can be obtained during magnetic stimulation, the time involvement to collect > 15 replicates while ensuring adequate neuromuscular recovery, may greatly impact the efficacy of the measurement protocol and utility of the associated indices of performance.

The results of intra-session and inter-day comparisons, which also show that measurement precision during inter-day comparisons is significantly inferior by contrast to intra-session assessments, suggest the indices PF_V, EMD_V and EMD_E offer the greatest practical utility for the assessment of neuromuscular performance. In addition, indices of time to half peak force ($T_{½E}$, $T_{½V}$) should be the preferred criteria to estimate the force generating capabilities of the knee flexors.

Kinanthropometry:

Chapter 5

SINGLE MEASUREMENT RELIABILITY AND REPRODUCIBILITY OF VOLITIONAL AND MAGNETICALLY EVOKED INDICES OF NEUROMUSCULAR PERFORMANCE IN MALES AND FEMALES FOLLOWING AN ACUTE FATIGUE TASK

5.1 Abstract

Given the focus of current research highlighting the potential link between neuromuscular fatigue and injury (e.g. Gleeson et al., 1998; Mercer et al., 1998: Yeung et al., 1999), fatigue trials may form a integral part of any comprehensive clinical assessment protocol. However, there exists limited scrutiny of the measurement sensitivity characteristics of indices of neuromuscular performance following such interventions. The aim of this study was to examine the inter-day reproducibility and single measurement reliability of indices of voluntary and magnetically evoked neuromuscular performance in the knee flexors of males and females following an acute fatigue task. Assessments of volitional and magnetically evoked neuromuscular performance of the knee flexors of seven men (age: 29.6 ± 10.4 yrs; height 1.78 ± 0.04 m; mass 77.0 ± 7.7 kg (mean ± SD) and nine women (age 25.2 ± 4.2 yrs; height 1.69 ± 0.08 m; mass 62.8 ± 8.1 kg) of the preferred leg were obtained on three separate days immediately following an acute fatigue task. Inter-day assessment sessions were separated by at least three days. The exercise task required the participants to perform a fatigue trial of 30 seconds maximal static intermittent exercise of the knee flexors of the preferred leg. Inter-day measurement reproducibility was estimated by coefficient of variation (V%, adjusted for small-sample bias) and single measurement reliability by means of intra-class correlations (R_I) and standard error of a single measurement (SEM%) (95% confidence limits). The results showed there were no difference in measurement reproducibility between sexes and the technique of magnetic stimulation for the inter-day assessment of neuromuscular performance capacity offered equivocal measurement utility by comparison to traditional methods, except

for indices of peak force (P_TF_E). The current results indicate that single trial protocols do not offer sufficient measurement precision during inter-day assessments of neuromuscular performance following fatigue, either for intra-subject or intra-group comparisons. Most indices, however, were not associated with an increase in inter-day variability of performance following acute muscle fatigue and some indices ($T_{½E}$, EMD_E) showed superior measurement reproducibility ($p<0.01$). These results indicate that similar protocols can be used to assess individual inter-day performance under fatigued and un-fatigued conditions. In addition, judgements about the design of a measurement protocol and its associated level of measurement precision can be made on the same basis for both sexes. The current data suggest the indices PF_V and EMD_E offer the greatest practical utility for the assessment of post-fatigue neuromuscular performance based on the number of trials required to accurately discriminate intra-individual and intra-group performance differences (15 and 3 for PF_V, and 5 and 5 for EMD_E, respectively (95% confidence limits).

5.2 Introduction

The effectiveness of particular training or physical therapy interventions are frequently evaluated by means of inter-day assessments to monitor changes in performance capabilities (Hakkinen and Komi, 1983; Shelbourne and Gray, 1997; Gleeson et al., 1998a). While these comparisons can potentially offer integral information regarding individual performance, caution must be exercised during case-study investigations, since such demand stringent criteria for precision of measurement in order to make meaningful performance comparisons (Mercer and Gleeson, 2002). This may be of particular concern during inter-day assessments, since these may be associated with poorer measurement reliability characteristics compared to intra-session evaluations of individual neuromuscular performance (Gleeson and Mercer, 1996; chapter 4).

Magnetic stimulation has become increasingly used to estimate the performance capacity of the neuromuscular system (e.g. Barker et al., 1987; Evans et al., 1988; Zhu et al., 1992; Chokroverty et al., 1993; Polkey et al., 1996) due its ability to potentially counteract factors that confound the proper measurement of an individual's true maximal performance capacity during volitional muscular activation (Tsuji and Nakamura, 1988; Zhou et al., 1995; Hopkins and Ingersoll, 2000; Gleeson, 2001). However, only limited information is available in the contemporary scientific literature regarding the reproducibility and reliability characteristics of indices of neuromuscular performance associated with magnetic stimulation prior to fatigue (Polkey et al., 1996) and currently, no studies have investigated these issues following fatigue.

Given the focus of current research highlighting the potential link between neuromuscular fatigue and injury (e.g. Gleeson et al., 1998; Mercer et al., 1998: Yeung et al., 1999), fatigue trials may form a integral part of any comprehensive clinical assessment protocol. However, there exists limited scrutiny of the measurement sensitivity characteristics of indices of neuromuscular performance following such interventions. Currently, the 'best guess' of expected measurement error of indices of neuromuscular performance following acute muscle fatigue may thus be based on the typical decrease in absolute performance capabilities associated with such interventions (e.g. Gleeson et al., 1997; Horita and Ishiko, 1987; Zhou et al., 1996; Yeung et al., 1999) and the expected variability of performance measured prior to fatigue. Consequently, performance variation under conditions of muscle fatigue may be expected to be greater compared to un-fatigued conditions, despite constant technical and biological variability, since such will be expressed relative to reduced absolute performance levels. Based on the number of trials required to achieve an acceptable level of measurement sensitivity prior to fatigue (study 4.0), the utility of some indices of neuromuscular performance may be compromised, unless the fatiguing exercise intervention is associated with a reduction in biological variability.

Despite the results of the prior investigation showing equivalent measurement error between males and females, such should be re-evaluated following fatigue to investigate whether or not the reproducibility and reliability characteristics of neuromuscular performance scores differ systematically between sexes. Clearly any differences may impact the subsequent design of experimental protocols and ultimately measurement efficacy.

The aim of this study, therefore, is to examine the inter-day reproducibility and single measurement reliability of indices of voluntary and magnetically evoked neuromuscular performance in the knee flexors of males and females following an acute fatigue task.

5.3 Methods

5.3.1 *Participants*

Seven men (age: 29.6 (± 10.4) yrs; height 1.78 (± 0.04) m; body mass 77.0 (± 7.7) kg (mean [± SD]) and nine women (age 25.2 (± 4.2) yrs; height 1.69 (± 0.08) m; body mass 62.8 (± 8.1) kg) gave their informed consent to participate in this study. All participants were regularly involved in exercise (at least 3 times per week) and were asymptomatic at the time of assessment. Participants were instructed to refrain from strenuous physical activity for the 24 hours prior to the test. The assessment protocols were approved by the University of Wales, Bangor, Human Performance Ethics Review Committee. The time commitment of each participant within this study (inclusive of 4.0 and 6.0) approximated 2.5 hours.

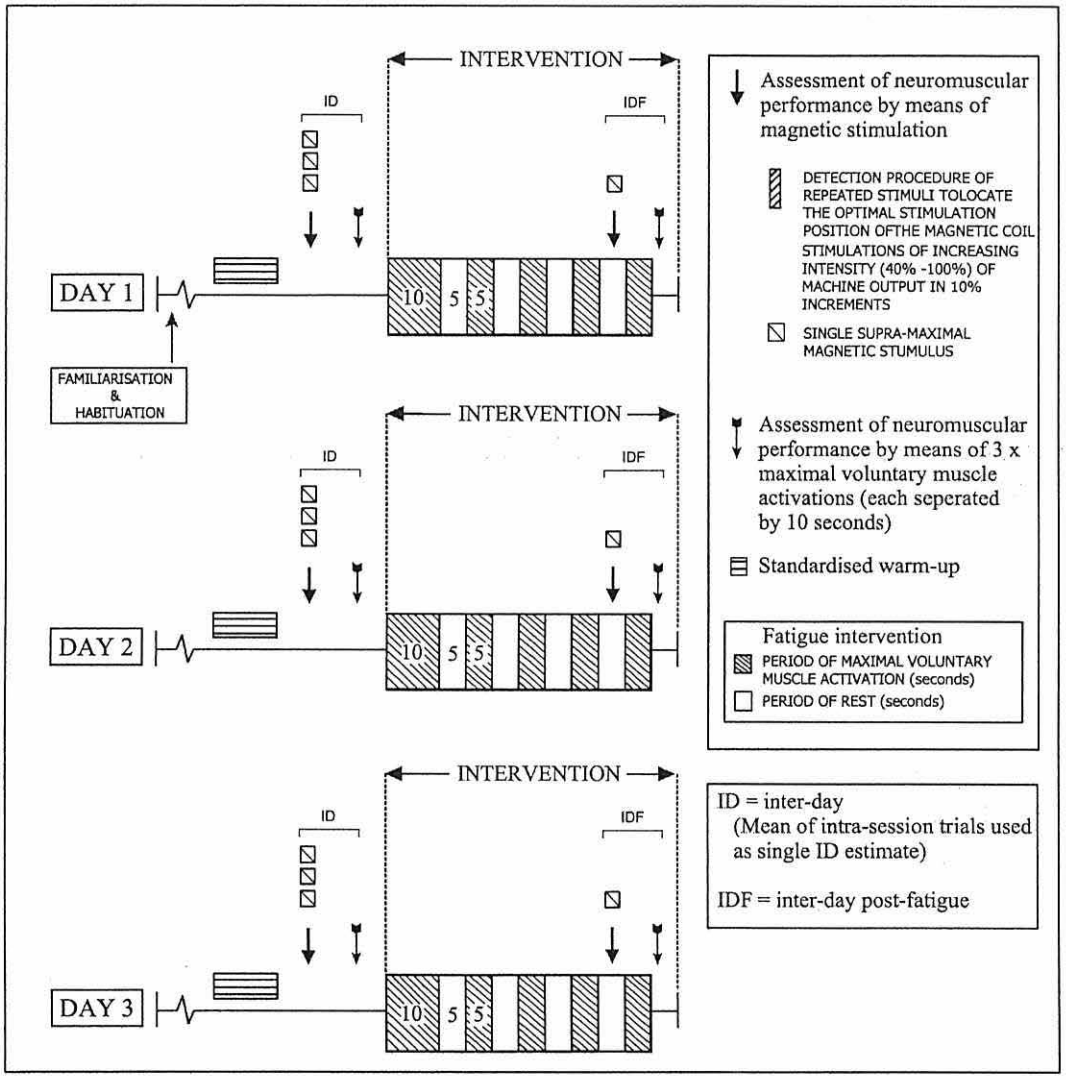
5.3.2 *Experimental procedures*

Following habituation procedures, participants completed a standardised warm-up consisting of five minutes cycle ergometry (90 Watts for males, 60 Watts for females) and a further five minutes of static stretching of the involved musculature prior to testing. Participants were then secured in a prone position on a custom-built dynamometer (modified from Gleeson et al., 1995). The evaluation of volitional and magnetically evoked neuromuscular performance of the knee flexors of the preferred leg were undertaken in accordance with the protocols and experimental conditions outlined in chapter 3.0 (sub-chapter 3.2), please see figure 3.1 for participant and dynamometer orientation (p 74).

Assessments of volitional and magnetically evoked neuromuscular performance of the knee flexors of the preferred leg were obtained on three separate days

immediately following an acute fatigue task. Inter-day assessment sessions were separated by at least three days. The fatiguing exercise task required the participants to perform a fatigue trial of 30 seconds maximal static intermittent exercise of the knee flexors of the preferred leg. Five bouts of maximal muscle activation were each separated by 5 seconds, the first episode of muscle activation lasted 10 seconds, followed by four further episodes of 5 seconds activation. Post-fatigue inter-day trends in absolute performance and measurement reproducibility and reliability were estimated by quantifying the performance variability associated with the three inter-day performance scores. Pre-fatigue inter-day data from the previous study (4.0) were included in the analyses for comparison. Participants were verbally encouraged during periods of maximal muscle activation. A description of the experimental protocol is shown in figure 5.1.

Figure 5.1. Schematic of the protocol for the assessment of inter-day measurement reproducibility and reliability for indices of volitional and magnetically evoked neuromuscular performance following an acute fatigue task.



5.3.3 *Indices of volitional neuromuscular performance*

The estimates of volitional neuromuscular performance of the knee flexors of the preferred leg were static peak force (PF_V), electromechanical delay (EMD_V), rate of force development (RFD_V) and time to half peak force ($T_{½V}$) and were defined and calculated in accordance with the methods outlined in chapter 3.0 (sub-chapter 3.5).

5.3.4 *Indices of magnetically evoked neuromuscular performance*

The estimates of magnetically evoked neuromuscular performance of the knee flexors of the preferred leg were static peak twitch force (P_TF_E), electromechanical delay (EMD_E), rate of force development (RFD_E) and time to half peak force ($T_{½E}$) and were defined and calculated in accordance with the methods outlined in chapter 3.0 (sub-chapter 3.6).

5.3.5 *Statistical Analyses*

The selected performance indicators were described using ordinary statistical procedures (mean (± SD)). The effect of the fatiguing exercise intervention was assessed for the index PF_V using a two (time: pre-fatigue; post-fatigue) by two (group: male; female) mixed-model analysis of variance (ANOVA) with repeated measures on the first factor. One-way repeated measures ANOVAs were used to check for systematic learning effects across trials within each session and between days (inter-day) for each index of performance. Magnetically evoked neuromuscular performance following fatigue was estimated on the basis of a single trial in order to minimise the time frame for potential recovery that would be associated with obtaining multiple estimates. Systematic examination of the data revealed that the standard deviation increased in proportion to the mean (heteroscedasticity). As such, coefficient of variation (V%), corrected for small sample bias (Sokal and Rohlf, 1981), was used to assess variability of indices across three inter-day trials. Coefficient of variation was calculated according to the expression (SD/mean) x (1+[1/4n] x 100) and expressed as a percentage, where n is the number of trials. The Shapiro-Wilks' test was used to check the normal distribution of V% scores. Single measurement reliability for each index was

assessed by computing intra-class coefficients (R_I) and standard error of a single measurement (SEM%) (95% confidence limits), expressed as a percentage of the group mean score according to the formula $((SD \times \sqrt{(1-R_I)})/mean) \times 100$ (multiplied by 1.96 to compute 95% confidence limits and assuming a normal distribution of scores). The Spearman-Brown prediction formula (Winer, 1981) was used to compute the expected reliability of the mean of multiple measurements for each index. Inter-day variability (V%) between groups (male, female) was compared using a two (time: pre-fatigue; post-fatigue) by two (mode of muscle activation: volitional; evoked) by two (group: males; females) mixed-model ANOVA with repeated measures on the first two factors. The assumptions underpinning the use of repeated measures ANOVA were checked and violations corrected by the Greenhouse-Geisser adjustment of the critical F-value, as indicated by $_{GG}$. Statistical significance was accepted at $p < 0.05$.

5.4 Results

One-way repeated measures ANOVAs revealed no significant differences in absolute scores across the three intra-session (volitional performance only) or three inter-day assessments for all indices of performance (PF_V, PF_E, EMD_V, EMD_E, RFD_V, RFD_E, $T_{½V}$ and $T_{½E}$), suggesting that changes in performance can be attributed to biological variation and technical error rather than systematic learning effects. Peak force data was subsequently amalgamated over the three inter-day measures to interrogate the effects of the fatiguing exercise task on PF_V. Table 5.1 shows the average absolute group mean performance scores for males and females prior to and immediately following the fatiguing exercise task. A significant (time by group) interaction associated with the mixed-model ANOVA ($F_{[1,14]} = 12.3$, $p < 0.01$) showed that the fatiguing exercise task elicited a reduction in absolute strength performance (PF_V) that was greater in males compared to females (265.1 (± 52.0) N vs. 311.8 (± 52.8) N [15.0% impairment] and 171.4 (± 33.9) N vs. 190.8 (± 48.6) N [10.2% impairment], respectively) (group mean score (± SD)).

Table 5.1. Absolute group mean (± SD) scores for indices of volitional ($_V$) and magnetically evoked ($_E$) neuromuscular performance for males and females amalgamated over the three inter-day measures.

Index		Pre-fatigue		Post-fatigue	
		Males	Females	Males	Females
PF_V	(N)	311.8 ± 52.8	190.8 ± 48.6	265.1 ± 52.0	171.4 ± 33.9
P_TF_E	(N)	14.7 ± 6.8	18.8 ± 6.5	12.9 ± 6.9	14.9 ± 5.0
EMD_V	(ms)	49.5 ± 6.4	50.6 ± 6.3	51.9 ± 13.1	61.9 ± 19.0
EMD_E	(ms)	32.2 ± 4.5	25.3 ± 2.9	27.1 ± 2.2	21.7 ± 1.4
RFD_V	($N.s^{-1}$)	2364.8 ± 530	1142.9 ± 242	1182.2 ± 523	659.4 ± 111
RFD_E	($N.s^{-1}$)	547.7 ± 287	678.1 ± 261	495.5 ± 313	599.8 ± 146
$T_{½V}$	(ms)	84.7 ± 15.6	117.8 ± 20.5	81.4 ± 13.2	100.2 ± 14.1
$T_{½E}$	(ms)	12.3 ± 2.5	11.4 ± 1.5	11.3 ± 1.5	9.9 ± 1.6

5.4.1 *Results of reproducibility analyses*

Table 5.2 shows group mean coefficient of variation (V%, adjusted for small sample bias), intra-class correlation coefficients (R_I) and standard error of a single measurement (SEM%) (95% confidence levels, expressed as a percentage of the mean group score) scores for inter-day day measures following the acute fatiguing exercise task for males and females separately. Repeated measures mixed-model ANOVAs of V% scores revealed no significant differences in variability between males and females for all the tested indices, data was therefore amalgamated to describe measurement precision. Table 5.3 shows group mean V%, R_I and SEM% scores for inter-day measures following the acute fatiguing exercise task (corresponding inter-day values prior to the fatigue intervention have also been included in table 5.3 for comparison).

Table 5.2. Inter-day post-fatigue group mean coefficient of variation (V% (mean ± SD)), intra-class correlation coefficient (R_I) and standard error of the measurement (SEM%) (95% confidence levels, expressed as a percentage of the mean group score) for indices of volitional ($_V$) and magnetically evoked ($_E$) neuromuscular performance for males and females.

Index	V%		R_I		SEM%	
	Males	Females	Males	Females	Males	Females
PF_V	8.7 ± 4.7	8.3 ± 4.5	0.81 ± 0.01	0.85 ± 0.01	10.5 ± 0.4	9.4 ± 0.6
P_TF_E	22.1 ± 14.4	31.5 ± 14.7	0.63	0.40	25.7	33.8
EMD_V	16.5 ± 5.3	10.8 ± 8.3	0.40 ± 010	0.85 ± 0.05	19.8 ± 3.0	14.3 ± 3.1
EMD_E	8.7 ± 6.0	6.2 ± 4.1	0.48	0.35	12.6	8.1
RFD_V	27.2 ± 7.9	31.2 ± 9.0	0.59 ± 0.10	0.37 ± 0.15	21.8 ± 7.0	31.6 ± 3.0
RFD_E	22.2 ± 13.1	30.7 ± 10.3	0.64	0.29	25.4	37.5
$T_{½V}$	13.7 ± 6.5	20.2 ± 7.1	0.61 ± 0.19	0.59 ± 0.04	13.1 ± 1.8	15.6 ± 6.4
$T_{½E}$	10.4 ± 4.5	13.8 ± 6.2	0.53	0.49	13.6	14.2

Table 5.3. Inter-day pre- and post-fatigue group mean coefficient of variation (V% (mean ± SD)), intra-class correlation coefficient (R_I) and standard error of the measurement (SEM%) (95% confidence levels, expressed as a percentage of the mean group score) for indices of volitional ($_V$) and magnetically evoked ($_E$) neuromuscular performance.

Index	Inter-day (pre-fatigue)			Inter-day (post-fatigue)		
	V%	R_I	SEM%	V%	R_I	SEM%
PF_V	8.9 ± 3.6	0.93 ± 0.02	8.7 ± 1.2	8.5 ± 5.0	0.91 ± 0.01	9.8 ± 0.3
P_TF_E	23.4 ± 7.6	0.73 ± 0.10	31.6 ± 9.4	27.4 ± 14.9	0.50	30.2
EMD_V	14.5 ± 5.5	0.64 ± 0.09	15.9 ± 3.1	17.3 ± 7.0	0.74 ± 0.02	18.7 ± 3.0
EMD_E	13.1 ± 6.3	0.51 ± 0.12	13.5 ± 1.9	7.3 ± 5.0	0.60	11.0
RFD_V	25.2 ± 9.0	0.71 ± 0.12	28.8 ± 5.0	29.5 ± 8.5	0.57 ± 0.08	30.2 ± 2.4
RFD_E	30.0 ± 11.4	0.70 ± 0.11	35.9 ± 8.7	27.0 ± 12.0	0.49	28.9
$T_{½V}$	15.8 ± 4.7	0.39 ± 0.13	16.2 ± 2.3	17.3 ± 7.4	0.43 ± 0.10	20.4 ± 2.5
$T_{½E}$	19.3 ± 5.2	0.32 ± 0.13	20.3 ± 4.5	12.3 ± 5.7	0.56	14.4

Indices of peak force (PF_V/P_TF_E)

A significant main effect for mode of muscle activation associated with the repeated measures mixed-model ANOVA ($F_{[1,14]} = 40.9$, $p < 0.001$) revealed greater variability of performance associated with magnetically evoked compared to volitional muscle activation (27.4 (± 14.9) % vs. 8.5 (± 5.0) % (group mean V% (± SD), respectively).

Indices of electromechanical delay (EMD_V/EMD_E)

A significant (time by mode of muscle activation) interaction associated with the repeated measures mixed-model ANOVA ($F_{[1,14]} = 15.2$, $p < 0.01$) revealed that while a small increase in inter-day variability of performance was observed following the acute fatiguing exercise task for EMD_V compared to pre-fatigue values (14.5 (± 5.5) % vs. 17.3 (± 7.0) %, group mean V% (± SD)), V% scores associated with the fatiguing exercise intervention for EMD_E were reduced (13.1 (± 6.3) % vs. 7.3 (± 5.0) %).

Indices of rate of force development (RFD_V/RFD_E)

Analyses revealed no significant interaction or main effects.

Indices of time to half force ($T_{½V}/T_{½E}$)

A significant (time by mode of muscle activation) interaction associated with the repeated measures mixed-model ANOVA ($F_{[1,14]} = 9.2$, $p < 0.01$) revealed that while a minor increase in inter-day variability of performance was observed following the acute fatiguing exercise task for $T_{½V}$ compared to pre-fatigue values (15.8 (± 4.7) % vs. 17.3 (± 7.4) %, group mean V% (± SD)), V% scores associated

with the fatiguing exercise intervention for $T_{½E}$ were reduced (19.3 (± 5.2) % vs. 12.3 (± 5.7) %).

5.4.2 *Results of single measurement reliability analyses*

The R_I for PF_V only exceeded a clinically acceptable reliability coefficient threshold of greater than 0.80 (Currier, 1984) during inter-day measures immediately following the fatiguing exercise task, however, SEM% scores (95% confidence limits) shows a limited capability to detect performance differences during intra-group comparisons.

5.5 Discussion

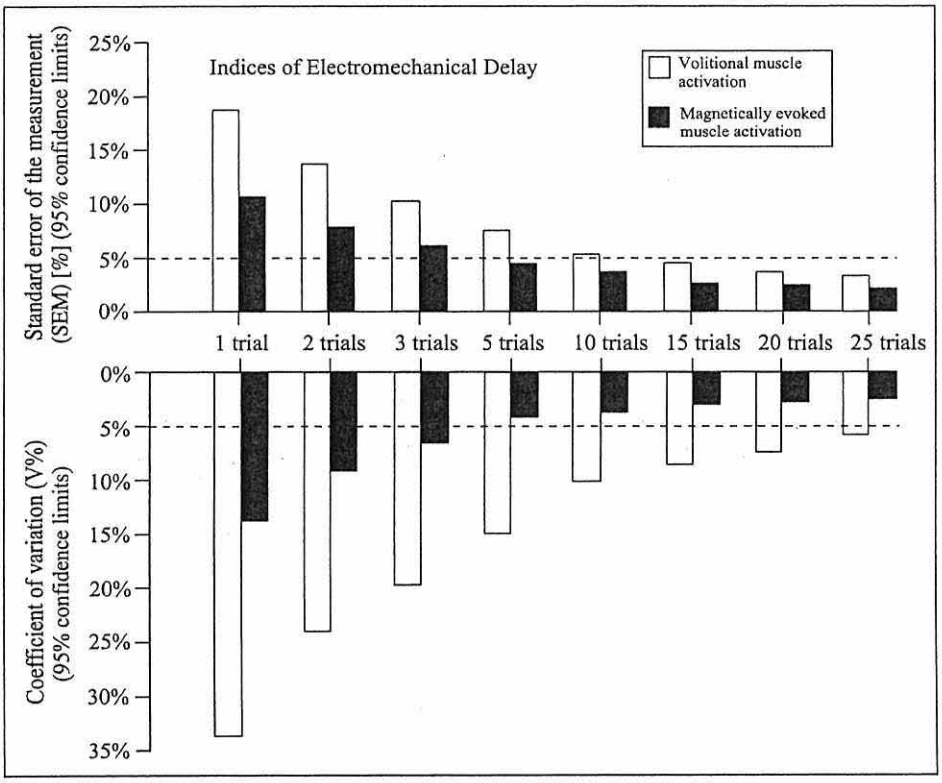
5.5.1 *Precision of measurement associated with inter-day estimates of performance following acute muscle fatigue*

The acute fatiguing exercise task induced fatigue in the knee flexors, characterised by a significant decrease in PF_V from pre- to post-fatigue levels. The average magnitude of PF_V performance decrement over the three days in the current study was 15.0% for males and 10.2% for females, which is congruent with the extent of performance loss associated with match play in team games such as soccer (Gleeson et al., 1998b).

The results show that the acute fatigue task was not associated with a significant change in the performance variability of indices PF_V, P_TF_E, RFD_V and RFD_E by comparison to pre-fatigue inter-day measures as assessed by coefficient of variation (adjusted for small sample size) (please see table 5.3). Despite this, the V% scores show that on the basis of a single trial these indices demonstrate a compromised capability to discriminate subtle changes in performance during intra-individual comparisons. Computation of 95% confidence limits using the central limit theorem (Thomas and Nelson, 1996), reveals a window of error of at best ±16.7%, for PF_V and at worst ±57.8% for RFD_V. While such performance changes are conceivable following injury, or during the early stages of a physical therapy programme (e.g. RFD_V of knee flexors: 720 $N.s^{-1}$ vs. 1836 $N.s^{-1}$ at 1.5 months and 3 months, respectively, during rehabilitation following ACL reconstructive surgery: ~ 61% change in performance [unpublished data]), higher levels of conditioning may demand more stringent levels of measurement precision. Clearly, these results indicate that on the basis of a single measure, the detection of only relatively gross

intra-individual changes in knee flexor neuromuscular performance between days is possible, either in fatigued or un-fatigued conditions.

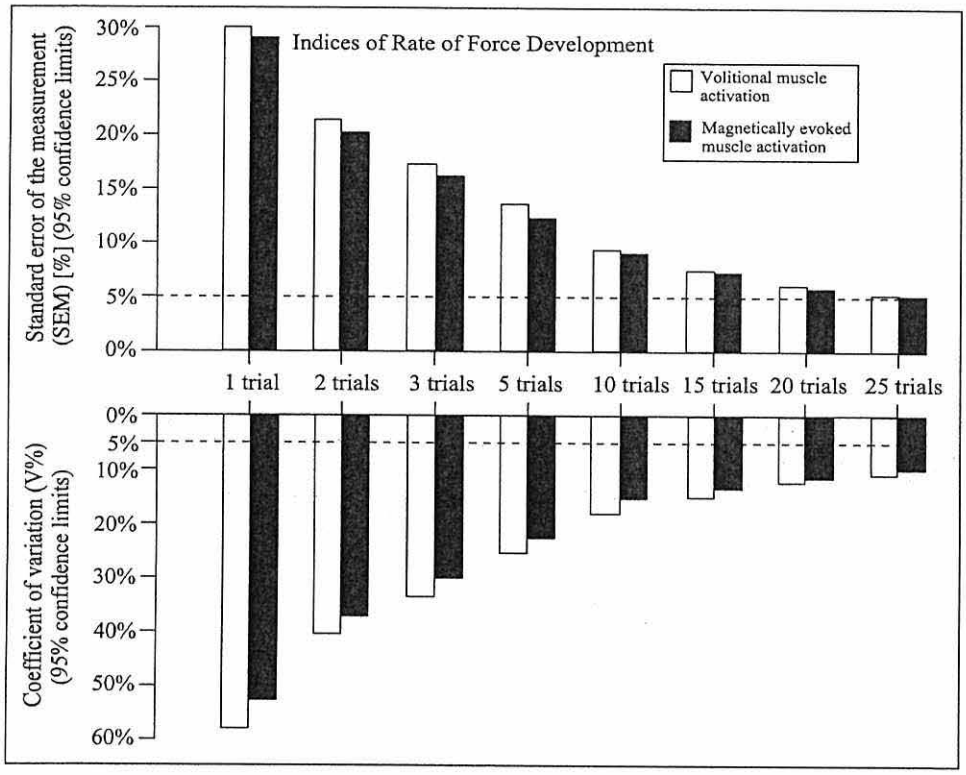
Figure 5.2. Error associated with the assessment of EMD_V (open bars) and EMD_E (closed bars) using 1 to 25 inter-day trials: coefficient of variation (V% [95% confidence limits]) and standard error of the measurement (SEM% [95% confidence limits]) following acute muscle fatigue.



The current fatigue protocol was associated with a small increase in variability of indices EMD_V and $T_{½V}$ yet, the V% scores of the equivalent magnetically evoked parameters (EMD_E, $T_{½E}$) were significantly reduced by comparison to inter-day assessment prior to fatigue (please see table 5.3). The physiologic underpinnings contributing to an improved consistency of these magnetically evoked parameters are currently unclear. However, the temporal response of a muscle, in particular EMD, is influenced considerably by the time taken to stretch the series elastic component (Cavanagh and Komi, 1979; Norman and Komi, 1979). Connective

tissue and muscle-tendon units subjected to a constant stress can elongate over time (stress-relaxation) effecting an increased length at a given load (Stone, 1992), in addition, strong static activation of muscle, as performed in the current protocol, can induce reactive hyperemia (McComas, 1996) potentially distending the knee flexor muscle group. These processes, which may substantively reduce the 'slack' within the system, might have contributed to the observed improvement in consistency of the temporal neuromuscular response. Such may be exclusively associated with magnetically evoked muscle activation, due to potentially better standardisation of recruitment of fast-twitch motor units by comparison to voluntary conditions (Garnett and Stephens, 1981; Benecke, 1996). Using the criterion that the estimated error of the mean score of multiple trials would be expected to vary inversely with the square root of the number of intra-individual replicates (assuming normal distribution) (Winer, 1981), the index EMD_E offers the greatest practical utility for intra-individual comparisons under conditions of muscle fatigue, requiring the mean score of 5 trials (95% confidence limits) to achieve an arbitrarily acceptable measurement error of better than 5% during inter-day assessments (please see figure 5.2). The equivalent threshold number of trials for indices PF_V, EMD_V, $T_{½V}$ and $T_{½E}$ to achieve a similar level of measurement precision is 15, >25, >25, and 25 (95% confidence limits), respectively. The number of required trials is greater still for indices P_TF_E, RFD_V and RFD_E (>28). These results clearly indicate that intra-individual neuromuscular performance under conditions of muscle fatigue cannot be properly evaluated on the basis of a single trial. Careful consideration should, therefore, be given to any intra-individual assessment under these conditions, particularly if such form the basis of important clinical judgements regarding of a safe return to match-play following a period of injury/rehabilitation.

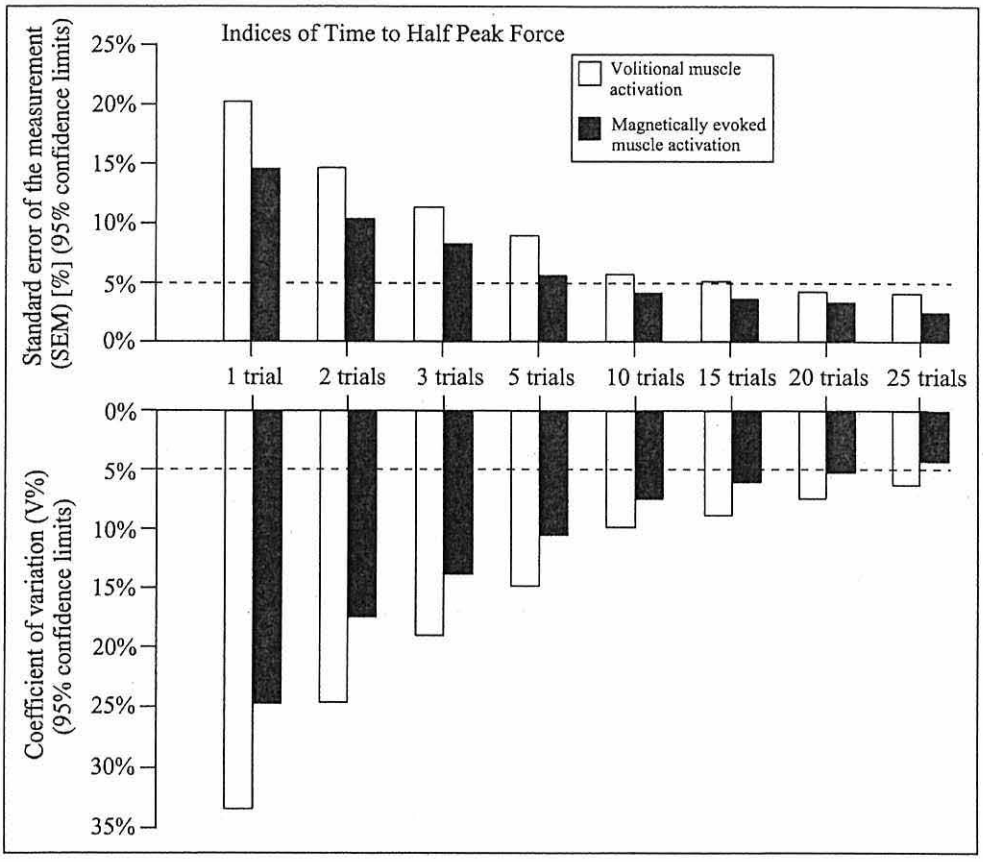
Figure 5.3. Error associated with the assessment of RFD_V (open bars) and RFD_E (closed bars) using 1 to 25 inter-day trials: coefficient of variation (V% [95% confidence limits]) and standard error of the measurement (SEM% [95% confidence limits]) following acute muscle fatigue.



Commensurate with the findings of the previous study (4.0), during inter-day measures the R_I for PF_V only (0.91 ± 0.01) exceeds a clinically acceptable reliability coefficient of greater than 0.80 (Currier, 1984). However, the group mean SEM% scores indicate a limited capability to detect performance differences associated with intra-group comparisons based on single-trial assessments (9.8 ± 0.3 %). The capability to discriminate subtle changes in neuromuscular performance would require a mean score of multiple trials as the basis for estimating performance in order to reduce measurement error (Gleeson et al., 2002). The Spearman-Brown prophecy formula (Winer, 1981) used in conjunction with the calculation of SEM% suggests that the indices PF_V and EMD_E (please see figure 5.2) offer the greatest practical utility following acute muscle fatigue during intra-group comparisons,

requiring the mean of 3 and 5 trials, respectively to achieve a level of measurement precision of ±5% or better. The equivalent threshold number of trials for indices $T_{½V}$, $T_{½E}$ and EMD_V are 20, 10 and 15, respectively. Based on the number of trials required to achieve an acceptable level of measurement precision (< ±5%), indices of time to half peak force ($T_{½V}$, $T_{½E}$) should be the preferred criterion over indices of rate of force development to estimate force generating capacity, since the latter indices (RFD_V, RFD_E) would require the mean score of >25 replicates to achieve an equivalent level of measurement error. Indeed, application of such neuromuscular assessment within certain populations, such as elite performance athletes, or patients approaching optimally rehabilitated status, may demand a level of measurement precision exceeding ±5%. During intra-group comparisons, the mean score of 5 and 15 trials would be required for indices PF_V and EMD_E, respectively in order to achieve a level of measurement error of ±3% or better (SEM%, 95% confidence limits). However, during intra-individual comparisons (V%, 95% confidence limits), such would be inflated to 35 and 25 trials, respectively, to achieve an equivalent level of measurement sensitivity. Consequently, if these parameters remain a key component during neuromuscular assessment, the clinician/researcher may have to accept a reduced level of measurement precision under conditions of acute muscle fatigue.

Figure 5.4. Error associated with the assessment of $T_{½V}$ (open bars) and $T_{½E}$ (closed bars) using 1 to 25 inter-day trials: coefficient of variation (V% [95% confidence limits]) and standard error of the measurement (SEM% [95% confidence limits]) following acute muscle fatigue.



4.5.2 *Summary*

Commensurate with the findings of the previous investigation (4.0), there were no differences in measurement reproducibility between sexes. In addition, the technique of magnetic stimulation for the inter-day assessment of neuromuscular performance capacity offered equivalent measurement utility by comparison to traditional methods, except for indices of peak force (PF_V, P_TF_E). As perhaps predicted from the results of the previous study (4.0), the current results indicate that single trial protocols do not offer sufficient measurement precision during inter-day assessments of neuromuscular performance following fatigue, either for intra-

subject or intra-group comparisons. Surprisingly, however, most indices were not associated with an increase in inter-day variability of performance following acute muscle fatigue, in fact, some indices ($T_{½E}$, EMD_E) were associated with superior measurement reproducibility. Accordingly, similar protocols can be used to assess individual inter-day performance prior to and following fatigue, though, consideration should be given to indices $T_{½V}$ and EMD_V where small increases in V% scores were observed. The current results suggest the indices PF_V and EMD_E offer the greatest practical utility for the assessment of post-fatigue neuromuscular performance based on the number of trials required to accurately discriminate intra-individual and intra-group performance differences (15 and 3 for PF_V, and 5 and 5 for EMD_E, respectively). The assessment of individual performance following fatigue, however, requires careful consideration. Despite the relative ease of obtaining multiple trials, particularly by means of magnetic stimulation, the time required to obtain these trials would be associated with a continued recovery and the subsequent evaluation of progressively improving absolute performance levels.

Interventions:

Chapter 6

EFFECTS OF AN ACUTE FATIGUING EXERCISE TASK ON THE VOLITIONAL AND MAGNETICALLY EVOKED NEUROMUSCULAR PERFORMANCE OF THE KNEE FLEXORS IN MALES AND FEMALES

6.1 Abstract

Females have been estimated to be at five to eight times greater risk of anterior cruciate ligament injury compared to male counterparts (Gray et al., 1985; Ireland et al., 1997). The fact that the majority of such injuries are sustained by non-contact aetiologies (Noyes et al., 1983; Rees, 1994) highlights the potentially important contributions of protective neuromuscular mechanisms. Given the suspected link between fatigue and injury (Gleeson et al., 1998; Mercer et al., 1998: Yeung et al., 1999), the aim of this study was to examine the effects of an acute (30 seconds) intermittent bout of maximal intensity static exercise on the voluntary and magnetically evoked neuromuscular performance in the knee flexors of males and females. Assessments of volitional and magnetically evoked neuromuscular performance of the knee flexors of the preferred leg of seven men (age: 29.6 ± 10.4 yrs; height 1.78 ± 0.04 m; body mass 77.0 ± 7.7 kg (mean ± SD) and nine women (age 25.2 ± 4.2 yrs; height 1.69 ± 0.08 m; body mass 62.8 ± 8.1 kg) were obtained were obtained prior to and immediately after two treatment conditions. The treatment conditions were: (i) an intervention condition that required participants to perform a fatigue trial of 30 seconds maximal static intermittent exercise of the knee flexors of the preferred leg, (ii) a control condition of equivalent duration to the intervention, consisting of no exercise. Each treatment condition was separated by 20 minutes. The results showed that the fatigue intervention was associated with a substantive reduction of the volitional force generating capabilities of the knee flexors which was greater in males compared to females (15.0%, 10.2%, respectively) ($p < 0.01$). While the index of time to half peak force ($T_{½V}$) was prolonged by equal amounts in males and females (>23%), impairment to rate of

force development (RFD_V) capabilities was greater in males compared to females (51.7% vs. 44.2%, respectively) ($p < 0.05$). In addition, electromechanical delay performance (EMD_V) was impaired exclusively in females (19.3%, $p < 0.05$). Given the protective capability of the active knee stabilisers may be substantively related to the reaction time of the neuromuscular system to imposed dynamic forces (Wojtys and Huston, 1996; Gleeson et al., 1998a; Mercer et al., 1998), the significant increase of EMD_V in females following acute muscle fatigue may be congruent with increased threat to knee joint stability, particularly in the presence of concomitant impairments to PF_V, RFD_V and $T_{½V}$. Despite the exercise intervention causing fatigue and impairment to indices of volitional neuromuscular performance, the ultimate physiological capacity of the neuromuscular system of the knee flexors, as measured by magnetic stimulation, was either preserved ($T_{½E}$), or potentiated (EMD_E, P_TF_E, RFD_E) by similar amounts in both males and females (21%, $p < 0.001$; >15%, $p < 0.05$; >22%, $p < 0.05$, respectively). Preservation and potentiation of some indices of magnetically evoked neuromuscular performance following fatigue in both males and females may suggest a vital compensatory mechanism and a capability to overcome the effects of impaired voluntary neuromuscular performance. Increased risk of ACL injury is likely to reflect the complex interaction of several factors, some of which may include neuromuscular conditioning, susceptibility to fatigue and ability to access the full capacity of the neuromuscular system at crucial times.

6.2 Introduction

A conceptual model that defines the normal limits of knee joint movement, depicts the interaction of putatively passive elements (osseous geometry, ligaments, menisci and capsular structures) with the active stabilisers (musculature) (Johansson et al., 1991; Fu et al., 1993). During strenuous activities, mechanical loading of the knee joint can often exceed the tensile capacities of the passive structures (Johansson et al., 1991). As a consequence, greater reliance may be placed on the protective capabilities of the surrounding musculature in order to maintain joint integrity (Gleeson et al., 1998a). Optimal functioning of the active muscle stabilisers, in particular the knee flexors, is considered fundamental to the prevention of anterior cruciate ligament (ACL) injury (Johansson, 1991; Rees, 1994; Gleeson and Mercer, 1996). The ACL is the primary restraint to anterior tibial translation relative to the femur (Fu et al., 1993; Rees, 1994) and is the most commonly injured of all the knee ligaments (Rees, 1994; Bollen, 1998). Accumulating evidence of an ACL injury epidemic by means of non-contact mechanisms in team sports athletes (Rees, 1994; Hutchinson and Ireland, 1995; Ireland et al., 1997) underscores the potentially important contribution of neuromuscular mechanisms to the maintenance of dynamic joint stability and the avoidance of injury. The protective capability of the active stabilisers may not be determined wholly by the capacity of the musculature to produce large amounts of force. Recent research has indicated the possibility that the reaction time of the neuromuscular system to imposed dynamic forces may be equally important to the protection of the joint system (Wojtys and Huston, 1996; Gleeson et al., 1998a; Mercer et al., 1998). One aspect of the overall neuromuscular reaction time has

been defined as electromechanical delay (EMD) and represents the time between the onset of electrical activity and the onset of tension in skeletal muscle (Norman and Komi, 1979; Zhou et al., 1996). This index of performance has been recognised as a potentially influential factor in knee joint stability (Gleeson et al., 1997; Gleeson et al., 1998a; 1998b; Mercer et al., 1998; Gleeson et al., 2000).

Acute muscle fatigue induced by means of maximal voluntary muscle activation (MVMA) has the potential to cause dramatic increases in EMD of between 42% to 70% longer compared with pre-fatiguing exercise values (Horita and Ishiko, 1987; Zhou et al., 1996). Clearly, any temporal impairment of this type to the volitional capability of the muscle stabilisers, coupled with concomitant decreases in the capacity for generating peak force may affect the timely harnessing of forces facilitating dynamic joint stabilisation and place the sports performer at increased risk of injury. In contrast, episodes of acute fatiguing exercise involving sub-maximal intensities may offer minimal physiological perturbance to the performance capability of the neuromuscular system and its capacity for joint protection. For example, studies that have used protocols involving sub-maximal fatiguing exercise intensity fatiguing tasks (70% and 50% of static MVMA, respectively) have reported no impairment to EMD performance (van Dieen et al., 1991; Vos et al., 1991).

Traditionally, volitional activation of muscle has been used to estimate neuromuscular performance capability. However, factors that might negatively influence performance consciously or sub-consciously such as waning motivation, injury and associated neuromuscular inhibition, potentially confound the proper

measurement of an individual's true maximal performance capacity under such circumstances (Hopkins and Ingersoll, 2000; Gleeson, 2001). Recent technological advances have enabled the evoked assessment of the neuromuscular system by means of magnetic stimulation of a motor nerve. This is a relatively painless process and has the capability to activate the fastest motor units (Maertens de Noordhout, 1991). As such, it offers an insight into the true maximal physiological performance capacity of the neuromuscular system (King and Chippa, 1989).

Although no studies have yet investigated the effects of acute muscle fatigue on the magnetically evoked EMD of the knee flexors, scrutiny of fatigue-related changes to EMD measured using electrically evoked activation of muscle have yielded conflicting findings of impaired (Zhou, 1996) and unchanged performance (Strojnik and Komi, 1998). Some investigators have also reported potentiated performance, defined as a significant decrease in EMD from pre- to post-fatigue scores (Sahlin and Seger, 1995).

The importance of elucidating fully processes that may compromise neuromuscular performance may be amplified when considering the dynamic stabilisation of the knee joint in the female athlete. There is accumulating evidence to show that females are five to eight times more likely to injure their ACL compared to male counterparts given equivalent exposure to sport (Gray et al., 1985; Hutchinson and Ireland, 1995; Arendt and Dick, 1995; Ireland et al., 1997). While no clear evidence exists to fully explain this phenomenon, there is consensus that increased risk of ACL injury in females may be associated with factors including differences in lower extremity alignment and exaggerated hormonal influences on tissue

compliance compared to males (Hutchinson and Ireland, 1995; Wojtys et al., 1998; Harmon and Ireland, 2000). In addition, there is some suggestion of sex-linked differences in temporal parameters associated with volitional activation of un-fatigued muscle, whereby females have demonstrated prolonged EMD values compared to male counterparts (Winter and Brookes, 1991).

Increased threat to knee joint integrity is likely to reflect a complex interaction of passive and active structures, which may vary under conditions of muscle fatigue. To date, there has been no investigation into the neuromuscular performance capacities of the knee flexors as assessed by magnetic stimulation and the effects of acute muscle fatigue in males and females.

The aim of this study, therefore, is to examine the effects of an acute (30 seconds) intermittent bout of maximal intensity static fatiguing exercise on the voluntary and magnetically evoked neuromuscular performance in the knee flexors of males and females.

6.3 Methods

6.3.1 *Participants*

Seven men (age: 29.6 (± 10.4) yrs; height 1.78 (± 0.04) m; body mass 77.0 (± 7.7) kg (mean [± SD]) and nine women (age 25.2 (± 4.2) yrs; height 1.69 (± 0.08) m; body mass 62.8 (± 8.1) kg) gave their informed consent to participate in this study. All participants were regularly involved in fatiguing (at least 3 times per week) and were asymptomatic at the time of assessment. Participants were instructed to refrain from strenuous physical activity for the 24 hours prior to the test. The assessment protocols were approved by the University of Wales, Bangor, Human Performance Ethics Review Committee. The time commitment of each participant within this study (inclusive of 4.0 and 5.0) approximated 2.5 hours.

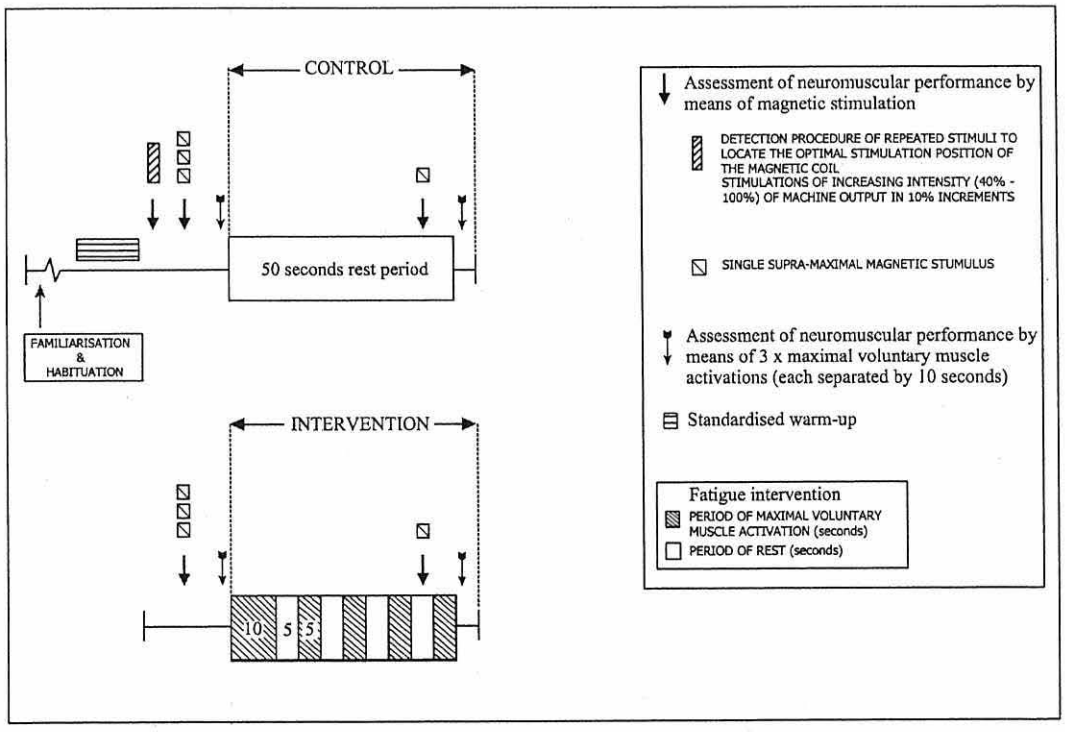
6.3.2 *Experimental procedures*

Following habituation procedures, participants completed a standardised warm-up consisting of five minutes cycle ergometry (90 Watts for males, 60 Watts for females) and a further five minutes of static stretching of the involved musculature prior to testing. Participants were then secured in a prone position on a custom-built dynamometer (modified from Gleeson et al., 1995). The evaluation of volitional and magnetically evoked neuromuscular performance of the knee flexors of the preferred leg were undertaken in accordance with the protocols and experimental conditions outlined in chapter 3.0 (sub-chapter 3.2), please see figure 3.1 for participant and dynamometer orientation (p 74).

Assessments of volitional and magnetically evoked neuromuscular performance of the knee flexors of the preferred leg were obtained prior to and immediately after

two treatment conditions, separated by 20 minutes: (i) an intervention condition that required participants to perform a fatigue trial of 30 seconds maximal static intermittent fatiguing exercise of the knee flexors of the preferred leg (There were five bouts of maximal muscle activation in total, each separated by 5 seconds. The first bout of muscle activation lasted 10 seconds, followed by four further bouts of 5 seconds activation) (ii) a control condition of equivalent duration to the intervention, consisting of no exercise. The control condition was performed first in order to avoid any potential carry-over effects of fatigue. Participants were verbally encouraged during periods of maximal muscle activation. A description of the experimental protocol is shown in figure 6.1.

Figure 6.1. Schematic of the protocol for the assessment of the effects of an acute fatiguing exercise task on the volitional and magnetically evoked neuromuscular performance of the knee flexors.



6.3.3 *Indices of volitional neuromuscular performance*

The estimates of volitional neuromuscular performance of the knee flexors of the preferred leg were static peak force (PF_V), rate of force development (RFD_V), time to half peak force ($T_{½V}$) and EMD (EMD_V) and were defined and calculated in accordance with the methods outlined in chapter 3.0 (sub-chapter 3.5).

6.3.4 *Indices of magnetically evoked neuromuscular performance*

The estimates of magnetically evoked neuromuscular performance of the knee flexors of the preferred leg were static peak twitch force (P_TF_E), rate of force development (RFD_E), time to half peak force ($T_{½E}$) and electromechanical delay (EMD_E) and were defined and calculated in accordance with the methods outlined in chapter 3.0 (sub-chapter 3.6).

6.3.5 *Statistical analysis*

The selected performance indicators were described using ordinary statistical procedures (mean ± SD). The effect of the fatiguing exercise intervention was assessed for each index of performance using separate two (condition: control; fatigue) by two (time: pre; post) by two (group: male; female) mixed-model ANOVAs with repeated measures on the first two factors. The assumptions underpinning the use of repeated measures ANOVA were checked and violations corrected by the Greenhouse-Geisser adjustment of the critical F-value, as indicated by $_{GG}$. Statistical significance was accepted at $p < 0.05$.

The experimental design offered an approximate .80 power of avoiding a Type-II error when employing a least detectable difference of 16 N, 8 ms, 350 $N.s^{-1}$ and 12

ms for PF_V, EMD_V, RFD_V and $T_{½V}$ respectively and 3.13 N, 3.5 ms, 208 N. s^{-1} and 1.1 ms for P_TF_E, EMD_E, RFD_E and $T_{½E}$ respectively.

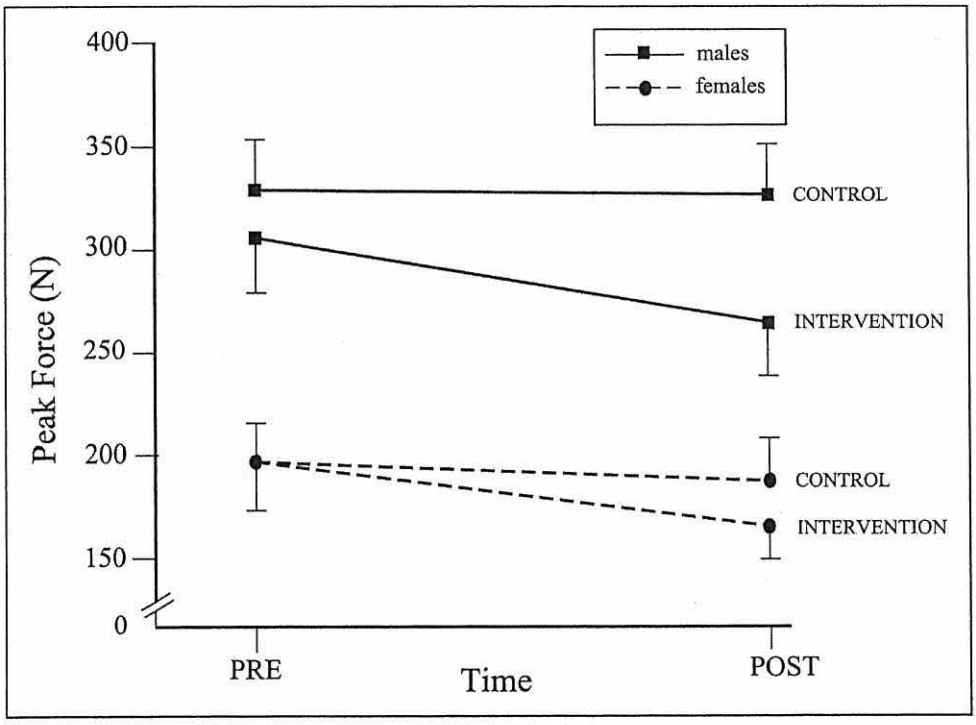
6.4 Results

6.4.1 *Volitional muscle activation*

Indices of volitional peak force (PF_V)

A significant three-factor interaction associated with the mixed-model ANOVA showed that while absolute strength performance was preserved during the control task, the fatiguing exercise task elicited a reduction in absolute strength performance in both males and females ($F_{[1,14]} = 14.0$, $p < 0.05$). However, the absolute strength performance (group mean score (± SD)) was impaired to a greater extent in males than in females compared to baseline scores (265.1 (± 52.0)N vs. 311.8 (± 52.8)N [15% impairment] and 171.4 (± 33.9)N vs. 190.8 (± 48.6)N [10.2% impairment], respectively) (please see figure 6.2).

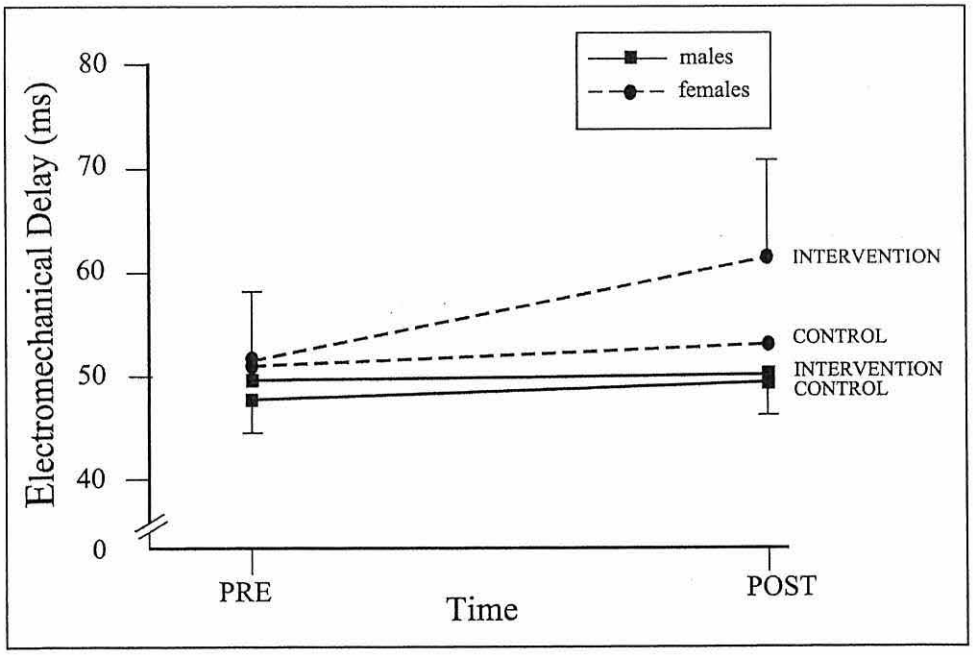
Figure 6.2. The effects of the fatiguing exercise protocol on the volitional peak force performance (PF_V) of the knee flexors (group mean ± SD).



Indices of volitional electromechanical delay (EMD_V)

A significant three-factor interaction associated with the mixed-model ANOVA ($F_{[1,14]} = 5.9$, $p < 0.05$) suggested that EMD_V performance was maintained during the control task for both groups and in the experimental condition for males. However, the fatiguing exercise task elicited a 19.3% reduction in EMD_V performance compared to baseline levels in females (61.9 (± 19.0)ms vs. 51.9 (± 13.1)ms, respectively) (please see figure 6.3). A-priori Helmert contrasts between group mean scores for males and females at baseline revealed no significant differences in EMD_V performance.

Figure 6.3. The effects of the fatiguing exercise protocol on the volitional electromechanical delay performance (EMD_V) of the knee flexors (group mean ± SD) (some SD bars removed for clarity).



Indices of volitional rate of force development (RFD_V)

A significant three-factor interaction associated with the mixed-model ANOVA showed that while absolute RFD_V was preserved during the control task, the

fatiguing exercise task elicited a reduction in RFD_V in both males and females ($F_{[1.14]} = 6.4$, $p < 0.05$). The absolute RFD_V performance (group mean score ± SD) was impaired to a greater extent in males than in females compared to baseline scores (1143 (± 243)$N.s^{-1}$ vs. 2364 (± 531)$N.s^{-1}$ [51.7% impairment] and 659 (± 111)$N.s^{-1}$ vs. 1182 ± (523)$N.s^{-1}$ [44.2% impairment], respectively).

Indices of volitional time to half peak force ($T_{½V}$)

A significant two-factor interaction associated with the mixed-model ANOVA showed that while absolute $T_{½V}$ performance was preserved during the control task, the fatiguing exercise task elicited a similar level of reduction (>23%) in $T_{½V}$ performance in both males and females compared to baseline scores (118 (± 21)ms vs. 85 (± 16)ms and 100 (± 14)ms vs. 81 (± 13)ms, respectively: $F_{[1,14]} = 40.8$, $p < 0.001$).

Table 6.1. Group mean (± SD) scores for indices of volitional neuromuscular performance for pre- and post-fatiguing exercise.

Index		Males		Females	
		Pre	Post	Pre	Post
PF_V	(N)	311.8 ± 52.8	265.1 ± 52.0	190.8 ± 48.6	171.4 ± 33.9
EMD_V	(ms)	49.5 ± 6.4	50.6 ± 6.3	51.9 ± 13.1	61.9 ± 19.0
RFD_V	($N.s^{-1}$)	2364 ± 530	1142 ± 242	1182 ± 523	659 ± 111
$T_{½V}$	(ms)	84.7 ± 15.6	117.8 ± 20.5	81.4 ± 13.2	100.2 ± 14.1

6.4.2 *Magnetically evoked muscle activation*

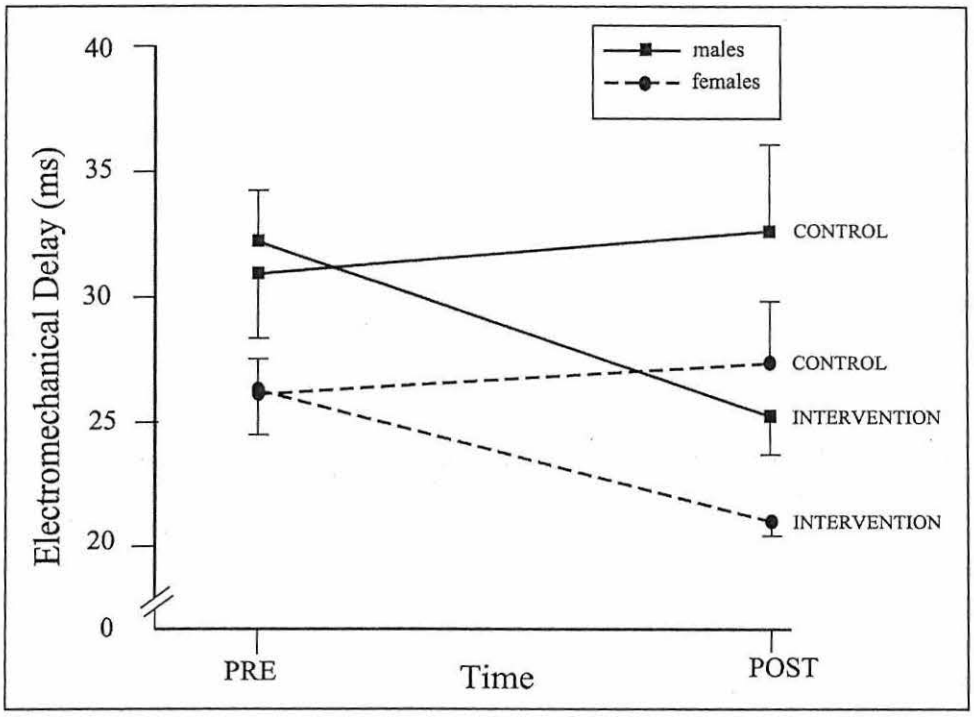
Indices of magnetically evoked peak twitch force (P_TF_E)

A significant two-factor interaction associated with the mixed-model ANOVA showed that while absolute P_TF_E performance was preserved during the control task, the fatiguing exercise task elicited a similar degree of potentiation (>15%) in P_TF_E performance in both males and females compared to baseline scores (14.7 (± 6.8)N vs. 18.8 (± 6.5)N and 12.9 (± 6.9)N vs. 14.9 (± 5.0)N, respectively: $F_{[1,14]} = 5.9$, $p < 0.05$).

Indices of magnetically evoked electromechanical delay (EMD_E)

A significant two-factor interaction associated with the mixed-model ANOVA showed that while absolute EMD_E performance was preserved during the control task, the fatiguing exercise task elicited a potentiation (21% decrease) in EMD_E latencies in both males and females ($F_{[1,14]} = 27.3$, $p < 0.001$). A-priori Helmert contrasts between males and females at baseline revealed significantly shorter absolute EMD_E values in females compared to males ($F_{[1,14]} = 7.3$, $p < 0.05$) (please see figure 6.4).

Figure 6.4. The effects of the fatiguing exercise protocol on the magnetically evoked electromechanical delay performance (EMD_E) of the knee flexors (group mean ± SD).



Indices of magnetically evoked rate of force development (RFD_E)

A significant two-factor interaction associated with the mixed-model ANOVA showed that while absolute RFD_E performance was preserved during the control task, the fatiguing exercise task elicited a similar degree of potentiation (>22%) in RFD_E performance in both males and females compared to baseline scores (547 (± 287)$N.s^{-1}$ vs. 678 (± 261)$N.s^{-1}$ and 495 (± 313)$N.s^{-1}$ vs. 600 (± 146)$N.s^{-1}$, respectively: $F_{[1,14]} = 4.7$, $p < 0.05$).

Indices of magnetically evoked time to half peak twitch force ($T_{½E}$)

The mixed-model ANOVA showed no significant interactions or main effects indicating that $T_{½E}$ performance was preserved during the control and fatiguing exercise treatment conditions in both males and females.

Table 6.2. Group mean (± SD) scores for indices of magnetically evoked neuromuscular performance for pre- and post-fatiguing exercise.

Index		Males		Females	
		Pre	Post	Pre	Post
P_TF_E	(N)	14.7 ± 6.8	18.8 ± 6.5	12.9 ± 6.9	14.9 ± 5.0
EMD_E	(ms)	32.3 ± 4.5	25.3 ± 2.9	27.1 ± 2.2	21.7 ± 1.4
RFD_E	($N.s^{-1}$)	547 ± 287	678 ± 261	495 ± 313	599 ± 146
$T_{½E}$	(ms)	12.3 ± 2.5	11.4 ± 1.5	11.3 ± 1.5	9.9 ± 1.6

6.5 Discussion

6.5.1 *Volitional neuromuscular performance*

The results show the fatiguing exercise intervention induced fatigue in the knee flexors, characterised by a significant decrease in PF_V from pre- to post-fatiguing exercise levels. The magnitude of PF_V performance decrement observed in the current study (15% for males and 10% for females) is congruent with the extent of performance loss associated with match play in team games such as soccer (Gleeson et al., 1998b). Furthermore, there was significant impairment of $T_{½V}$ performance that was similar for males and females (> 23%) and diminished RFD_V capability that was greater in males compared to females (51.7% and 44.2%, respectively). Such substantive impairments to the temporal force characteristics of the knee flexors observed in the current study, together with corroborating findings from other studies (e.g. Zhou et al., 1996; Gleeson et al., 1997) may suggest a reduced capability of the dynamic stabilisers to provide timely corrective responses to mechanical loading of the knee. The importance of these impairments to performance may be amplified particularly at knee angles where key ligamentous structures are already under greatest mechanical strain. Epidemiological research of ACL injury, however, shows that females are at increased risk compared to males given equivalent exposure to sport (e.g. Ireland and Wall, 1990). As such, this may suggest that factors other than PF_V and RFD_V performance capabilities, where males experienced larger impairments compared to females, have a greater influence on knee joint stability.

Despite males experiencing larger decrements to indices PF_V and RFD_V, an increase in EMD_V from pre- to post-fatigue levels (19.3%) was observed exclusively in

females. This is demonstrable of further impairments to the temporal capability of the knee flexors to produce force and which may be associated strongly with knee injury avoidance capabilities (Mercer et al., 1998). The processes involved in the conversion of excitation into muscle force can potentially contribute to the fatigue related changes in the force generating capability observed in the current study. These include the impairment of neuromuscular propagation caused by potassium ion (K^+) efflux from the muscle cells (Balog and Fitts, 1996; West et al., 1996) and changes in the intracellular milieu, reducing the amount of calcium ion (Ca^{2+}) release, sensitivity and the speed of cross bridge cycling (Metzger and Fitts, 1987; Fitts, 1996; Kent-Braun, 1999; Yeung et al., 1999). Yet, it is possible that other mechanisms may have contributed to the discriminate changes to the temporal characteristics of neuromuscular performance between males and females. Cavanagh and Komi (1979) proposed that the majority of the EMD is determined by the time taken to stretch the series elastic component (SEC), most of which is situated at the connective tissue attachments at the end of the muscle fibres (McComas, 1996). Muscle temperature increases associated with fatiguing exercise can directly cause an increase in compliance of the SEC (Zhou et al., 1998) and thus increases in EMD_V. Accordingly, increases in EMD_V might then be assumed in both males and females. The differential changes in EMD_V performance between sexes in the current study could be partially explained by the influence of female-specific hormones speculated to cause greater compliance in biologic tissue (Wojtys et al., 1998; Harmon and Ireland, 2000). Indeed, some investigations that have reported a longer EMD in females compared to male counterparts in un-fatigued muscle have proposed this as a potential cause (Bell and Jacobs, 1986; Winter and Brookes 1991). Instances where sex-linked differences in EMD_V performance are

not found (Morris and Beaudet, 1980; Houston et al., 1988 and the current study) may suggest potential compensatory mechanisms. For example, the many injury risk factors contended by females coupled with habituated exposure to scenarios where knee joint stability may be under threat might actually condition the neuromuscular system of the healthy athlete at functional joint angles. The subsequent formation of pre-programmed responses that can provide fast compensatory reactions to joint perturbations (Latash, 1998), may enable quick harnessing of the SEC, stiffening the joint in response to mechanical loading. This may be substantiated in the current study by the significantly quicker EMD_E of females compared to males observed prior to fatiguing exercise. Under conditions of muscle fatigue, however, this protective capability may be diminished. A reduction of the effectiveness of the fastest most powerful motor units as a consequence of fatigue could have impaired the temporal capability of the muscle to 'gather in' a potentially more compliant elastic tissue and have contributed to the longer EMD_V observed in females.

The substantive decrement to the force generating capacity of the knee flexors in males and females following acute fatigue may demonstrate a reduced capability to provide adequate dynamic restraint in response to mechanical loading of the knee joint. In addition, the concomitant increase in EMD_V in females may affect the timely harnessing of forces at the knee and place the female sports performer at increased risk of injury compared to males. Reference to neuromuscular performance evoked by magnetic stimulation may subsequently reveal whether these volitional performance decrements are associated with a reduction in the absolute capacity of neuromuscular system.

6.5.2 *Magnetically evoked neuromuscular performance*

The results show that, despite the fatiguing exercise intervention causing fatigue and impairment to indices of volitional neuromuscular performance, the ultimate physiological capacity of the neuromuscular system of the knee flexors, as measured by magnetic stimulation, was either preserved ($T_{½E}$), or potentiated (EMD_E, P_TF_E, RFD_E) by similar amounts in both males and females.

The 21% potentiation of EMD_E performance following fatigue in males and females in the current study is similar to the 16% improvement of electrically evoked EMD of the knee extensors reported by Sahlin and Seger (1995) following prolonged exhaustive cycling. The apparent coexistence of fatigue of volitional performance and potentiation of evoked performance (EMD_E) could be attributable to several factors. Possible increases in speed of neuromuscular transmission as a result of muscle temperature changes may actually yield minimal influence, since the proportion of EMD accounted for by muscle fibre conduction velocity is thought to be relatively small compared to the lengthening of the SEC (Komi, 1979; Zhou et al., 1995). It is suggested that connective tissue and muscle-tendon units subjected to a constant stress elongate over time (stress-relaxation), eliciting an increased length at a given load (Stone, 1992). Furthermore, strong static activation of muscle, as performed in the current protocol, can induce reactive hyperemia (McComas, 1996), potentially distending the muscle. A possible reduction in compliance within the system induced by stress-relaxation and hyperemia may have contributed substantially to the potentiated post-fatigue EMD_E performance observed in the current study. However, due to the differential responses of EMD_V

and EMD_E following fatigue, the relative contributions of specific fatigue and potentiating mechanisms are currently unclear.

The potentiation of some indices of magnetically evoked neuromuscular performance may be commensurate with the potential to overcome the fatigue-related impairments of the volitional performance capabilities. However, access to the full capacity of large high threshold motor units seems to be restricted under voluntary conditions (Tsuji and Nakamura, 1988; Zhou et al., 1995). This is currently demonstrated by the longer latencies associated with EMD_V (e.g. 51.9 ms) compared to EMD_E (e.g. 27.1 ms) and the fact that fatigue induced by volitional activation of muscle did not impair magnetically evoked neuromuscular performance. These results also suggest that methods of assessment of performance capacity must be carefully considered, since utilisation of solely volitional means of assessment may predispose a gross underestimation of the true capacity of the neuromuscular system, as illustrated by EMD_E and EMD_V performance comparisons.

Several investigators explain potentiation of evoked neuromuscular performance by means of metabolic factors. This may be more appropriate when describing indices of performance that are influenced to a lesser extent by the taking up of the 'slack' within the system. Potentiation of P_TF_E and RFD_E by 20.9% and 24.6%, respectively could have been influenced substantially by an exercise-induced increase in sensitivity of muscle contractile proteins to Ca^{2+} (O'Leary et al., 1997). This heightened Ca^{2+} sensitivity effects an increase in the rate of cross-bridge attachment, potentiating post-fatigue sub-maximal (twitch) responses to beyond pre-

fatigue levels (O'Leary et al., 1997; Gabriel and Boucher, 1998; Hamada et al., 2000; Rassier and MacIntosh, 2000).

6.5.3 *Implications to the sports performer*

The results of magnetically evoked muscle activation of the knee flexors show the effects of acute muscle fatigue on the neuromuscular performance capacity were equivocal for males and females. In addition, it appears that females retain a similar, and perhaps temporally superior, baseline neuromuscular performance capacity compared to males (although it is acknowledged that factors such as leg mass have not been taken into account). These results challenge sex-linked differences in neuromuscular capacity as a likely factor predisposing ACL injury and perhaps suggest other factors are associated more strongly with increased risk of injury.

The net result following acute volitional muscle fatigue may be a 'reserve capacity' of unused motor units. Yet, such preservation and potentiation of neuromuscular performance capacity may be of limited use to the sports performer due to potential protective central and peripheral neuromuscular inhibitory mechanisms (Hopkins and Ingersoll, 2000) restricting volitional access to the full performance capacity of a muscle. Accordingly, the utility of a potential 'reserve' capacity is entirely dependent on whether this inhibition can be overcome by the actual sub-conscious emergency responses elicited subsequent to potentially threatening joint perturbations.

6.5.4 *Summary*

Volitional neuromuscular performance was substantively impaired following acute fatiguing exercise from 10% (for PF_V for females) to 51.7% (for RFD_V for males). Males experienced generally greater fatigue-related impairments to the volitional force characteristics of the knee flexors, however, EMD_V performance was impaired exclusively in females. The significant increase of EMD_V in females following acute muscle fatigue may be congruent with increased threat to knee joint stability, particularly in the presence of concomitant decreases in PF_V, RFD_V and increases in $T_{½V}$. Preservation and potentiation of some indices of magnetically evoked neuromuscular performance following fatigue in both males and females may suggest a capability to overcome the effects of impaired voluntary neuromuscular performance. Yet, the utility of a preserved performance capacity to the sports performer is entirely dependent on whether the neuromuscular strategies observed subsequent to magnetic stimulation can be replicated under non-evoked conditions. Increased risk of ACL injury is likely to reflect the complex interaction of several factors, some of which may include neuromuscular conditioning, susceptibility to fatigue and ability to access the full capacity of the neuromuscular system at crucial times.

Interventions:

Chapter 7

EFFECTS OF SERIAL FATIGUING TASKS AND ACUTE RECOVERY ON INDICES OF VOLITIONAL AND MAGNETICALLY EVOKED NEUROMUSCULAR PERFORMANCE OF THE KNEE FLEXORS IN FEMALES

7.1 Abstract

The aim of this study was to investigate the effects of four serial bouts of maximal static exercise and acute recovery on volitional and magnetically evoked indices of neuromuscular performance of the knee flexors in females. Assessments of volitional and magnetically evoked neuromuscular performance of the knee flexors of the preferred leg of twenty females (age: 21.3 ± 2.3 yrs; height 1.68 ± 0.06 m; body mass 68.0 ± 6.3 kg [mean ± SD]) were obtained prior to, at pre-determined periods during, immediately after and at 1, 3 and 6 minutes during recovery in each of two conditions. The two conditions consisted of: (i) a control condition (CON) of equivalent duration to the subsequent fatiguing task intervention, consisting of no exercise; (ii) a fatiguing task (FAT) that required participants to perform 4 bouts of 35 seconds maximal static exercise of the knee flexors of the preferred leg. The results showed that the serial exercise task induced cumulative decrease to the peak force (PF_V) capabilities of the knee flexors of up to 15.9% compared to pre-exercise levels ($p < 0.05$), which recovered to 96.6% of pre-exercise values following six minutes rest ($p < 0.05$). An associated 25.5% increase in EMD_V following the first episode of exercise ($p < 0.05$) was maintained throughout the remainder of the intervention and recovery. In addition, magnetically evoked peak twitch force was impaired following the fourth episode of fatiguing exercise (23.3%, $p < 0.05$) and had not recovered by the end of the period of assessment. Given that optimal functioning of the knee flexors is fundamental to the prevention of anterior cruciate ligament (ACL) injury (Johansson, 1991; Rees, 1994; Gleeson and Mercer, 1996), such fatigue-related impairment to the associated neuromuscular capabilities may place the female athlete at increased and prolonged risk of knee injury. While some

indices of neuromuscular performance were impaired, magnetically evoked temporal responses were potentiated (EMD_E, 10.0%; $T_{½E}$, 8.2%) following the first and second episodes of exercise, respectively, which was maintained up to the 6-minute recovery measure. Potentiation of the temporal capacity of the knee flexors may be a vital neuromuscular compensatory strategy to enable joint protection during periods of decreased volitional performance capabilities. Accordingly, in some circumstances, an episode of fatiguing exercise may actually facilitate rather than impede neuromuscular performance in the knee flexors and ultimately enhance the capability to resist joint injury.

7.2 Introduction

There is accumulating evidence of an ACL injury epidemic by means of non-contact mechanisms in team sport athletes that shows females to be at five to eight times greater risk by comparison to male counterparts (Gray et al., 1985; Rees, 1994; Arendt and Dick, 1995; Hutchinson and Ireland, 1995; Ireland et al., 1997). These data may highlight the importance of neuromuscular mechanisms to knee joint stability, especially in the female athlete. Specifically, the protective capability of the active muscle stabilisers to avoid knee injury may be associated substantively with the reaction time of the neuromuscular system to imposed dynamic forces (Wojtys and Huston, 1996), in particular EMD (Gleeson et al., 1997; Gleeson et al., 1998a; Mercer et al., 1998; Gleeson et al., 2000).

Team games such as basketball and soccer frequently demand that participants perform intermittent periods of sprinting (Bangsbo, 1994; McInnes et al., 1995). These may be associated with greater impairments to the neuromuscular performance capabilities by comparison to a single acute episode of maximal exercise. For example, Zhou et al. (1996) reported a 70.5% increase in the EMD of the knee extensors following four bouts of thirty seconds maximal sprint cycling, exceeding reported 11.5% to 42% increases in EMD following single episodes of exercise (Gleeson et al., 1997; Horita and Ishiko, 1987). To date, there has been no investigation into the effects of repeated episodes of maximal exercise on the volitional and magnetically evoked performance of the knee flexors in females. Such impairments to the dynamic knee stabilisers may be associated with a reduced temporal capability to harness forces facilitating dynamic joint stabilisation and

place the sports performer at increased risk of injury compared to single episodes of exercise. Observations from the prior study (chapter 6.0), however, suggest that the body may exhibit certain compensatory mechanisms, whereby the physiologic capacity of the neuromuscular system (as estimated by magnetically evoked indices of performance such as EMD_E RFD_E and P_TF_E) is improved following volitional muscle fatigue. Such processes may be associated with the potential to counter any increased susceptibility to injury. Yet, there is some evidence to suggest that prolonged exercise protocols induce impairment to the magnetically evoked neuromuscular performance capacity of the knee extensors (Polkey et al., 1996). Reductions in the performance capacity of the dynamic knee stabilisers may, at best, preclude any compensatory mechanisms, but at worst, be associated with a decreased capacity to preserve joint integrity during periods of mechanical loading.

Decrements to the performance capabilities of the dynamic joint stabilisers that remain following cessation of exercise may also predispose the sports performer to increased risk of injury during the ensuing period of recovery. It is possible that repeated episodes of maximal exercise be associated with a longer time course of recovery compared to a single bout. For example, an 11.5% increase in volitional EMD of the knee flexors following an acute exercise task required approximately three minutes to recover to within pre-exercise values (Gleeson et al., 1997). Serial episodes of maximal exercise of the knee extensors eliciting 49.5% and 19.8% increases in volitional and electrically evoked EMD, respectively, were, however, associated with a recovery time of up to ten minutes (Zhou, 1996). These potentially prolonged impairments to neuromuscular performance may render the sports performer at increased and sustained risk of injury by comparison to single

acute episodes of exercise. This may be of particular concern to the female athlete involved in team games such as basketball because this type of sport is characterised by repeated high joint loading from multiple rapid accelerations, decelerations and changes in direction (McInnes et al., 1995). To date there has been no systematic investigation into the effects of serial acute maximal exercise tasks and subsequent recovery on volitional or magnetically evoked indices of neuromuscular performance of the knee flexors in females.

The aim of this study is, therefore, to investigate the effects of four serial bouts of maximal static exercise and acute recovery on volitional and magnetically evoked indices of neuromuscular performance of the knee flexors in females.

7.3 Methods

7.3.1 *Participants*

Twenty females (age: 21.3 ± 2.3 yrs; height 1.68 ± 0.06 m; body mass 68.0 ± 6.3 kg [mean ± SD]) gave their informed consent to participate in this study. All participants were involved in team sports, participating at least 3 times per week and were asymptomatic at the time of assessment. Participants were instructed to refrain from strenuous physical activity for the 24 hours prior to the test. The assessment protocols were approved by the University of Wales, Bangor, Human Performance Ethics Review Committee. The time commitment of each participant within this study approximated 1 hour.

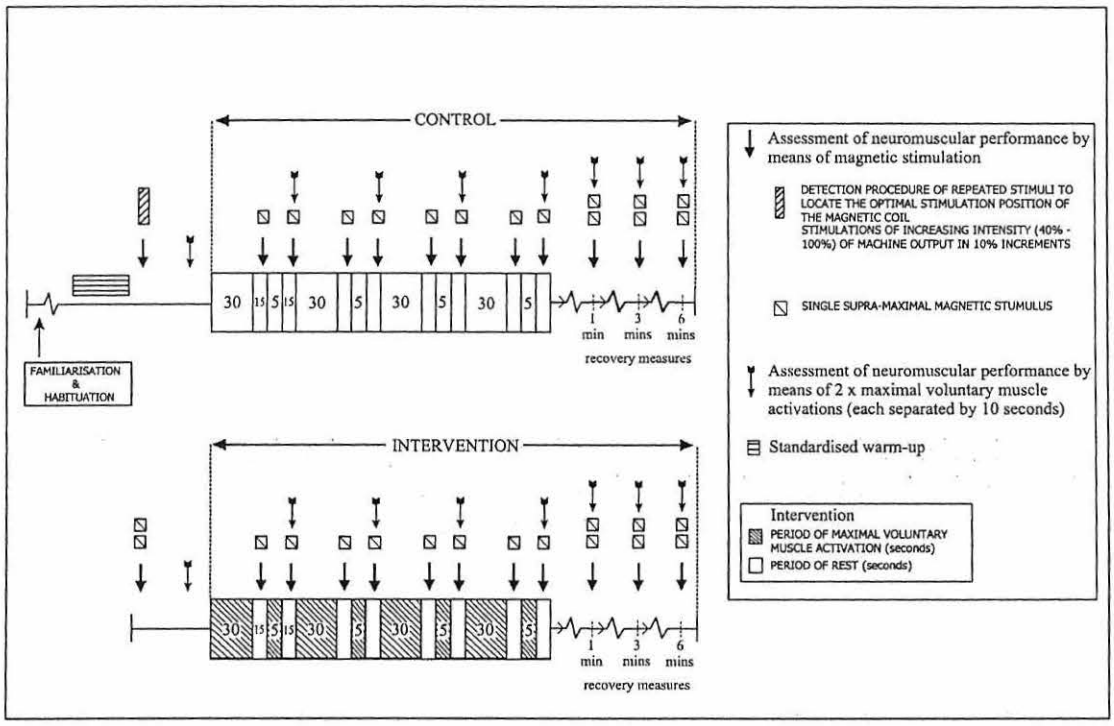
7.3.2 *Experimental procedures*

Following habituation procedures, participants completed a standardised warm-up consisting of five minutes cycle ergometry (60 Watts) and a further five minutes of static stretching of the involved musculature prior to testing. Participants were then secured in a prone position on a custom-built dynamometer (modified from Gleeson et al., 1995). The evaluation of volitional and magnetically evoked neuromuscular performance of the knee flexors of the preferred leg were undertaken in accordance with the protocols and experimental conditions outlined in chapter 3.0 (sub-chapter 3.2), please see figure 3.1 for participant and dynamometer orientation (p 74).

Assessments of volitional and magnetically evoked neuromuscular performance of the knee flexors of the preferred leg were obtained prior to ('pre'), at pre-determined periods during ('post1', 'post2', 'post3'), immediately after ('post4') and at 1, 3 and 6 minutes during recovery ('R1', 'R3', 'R6') in each of two

conditions. The two conditions were presented in sequence to minimise the potential intrusion of inter-day variability in performance measures in this experimental population and consisted of: (i) a control condition (CON) of equivalent duration to the subsequent fatiguing task intervention, consisting of no exercise; (ii) a fatiguing task (FAT) that required participants to perform 4 x 35 seconds maximal static exercise of the knee flexors of the preferred leg (each bout consisted of 30 seconds sustained effort followed by a 5 second rest period and a further bout of 5 seconds effort). Each 35-second bout of exercise was separated by approximately 15 seconds, during which time performance measures were obtained. The CON and FAT conditions were separated by at least 20 minutes. The inclusion of a control task will facilitate the estimation of the extent of intrusion from confounding factors such as systematic carry-over of the effects associated with the progression through intra- and inter-experimental conditions and assessment protocols. Participants were verbally encouraged during periods of maximal muscle activation. A description of the experimental protocol is shown in figure 7.1.

Figure 7.1. Schematic of the protocol for the assessment of the effects of serial fatiguing tasks and acute recovery on indices of volitional and magnetically evoked neuromuscular performance of the knee flexors.



7.3.3 *Indices of volitional neuromuscular performance*

The estimates of volitional neuromuscular performance of the knee flexors of the preferred leg were static peak force (PF_V), rate of force development (RFD_V), time to half peak force ($T_{½V}$) and EMD (EMD_V) and were defined and calculated in accordance with the methods outlined in chapter 3.0 (sub-chapter 3.5).

7.3.4 *Indices of magnetically evoked neuromuscular performance*

The estimates of magnetically evoked neuromuscular performance of the knee flexors of the preferred leg were static peak twitch force (P_TF_E), rate of force development (RFD_E), time to half peak force ($T_{½E}$) and electromechanical delay (EMD_E) and were defined and calculated in accordance with the methods outlined in chapter 3.0 (sub-chapter 3.6).

7.3.5 *Statistical analyses*

The selected performance indicators were described using ordinary statistical procedures (mean ± SD). The effect of the exercise intervention was assessed for each index of performance using separate two (condition: control; intervention) by five (time: pre; post1; post2; post3; post4) fully repeated measures ANOVAs. Recovery was investigated for each index of performance using separate two (condition: control; intervention) by four (post4; R1; R3; R6) fully repeated measures ANOVAs. *A priori* (reverse Helmert) contrasts were used to examine any significant differences between means. The assumptions underpinning the use of repeated measures ANOVA were checked and violations corrected by the Greenhouse-Geisser adjustment of the critical F-value, as indicated by $_{GG}$. Statistical significance was accepted at $p < 0.05$.

The experimental design offered an approximate .80 power of avoiding a Type-II error when employing a least detectable difference of 16 N, 8 ms, 350 $N.s^{-1}$ and 12 ms for PF_V, EMD_V, RFD_V and $T_{½V}$ respectively and 3.13 N, 3.5 ms, 208 $N.\ s^{-1}$ and 1.1 ms for P_TF_E, EMD_E, RFD_E and $T_{½E}$ respectively.

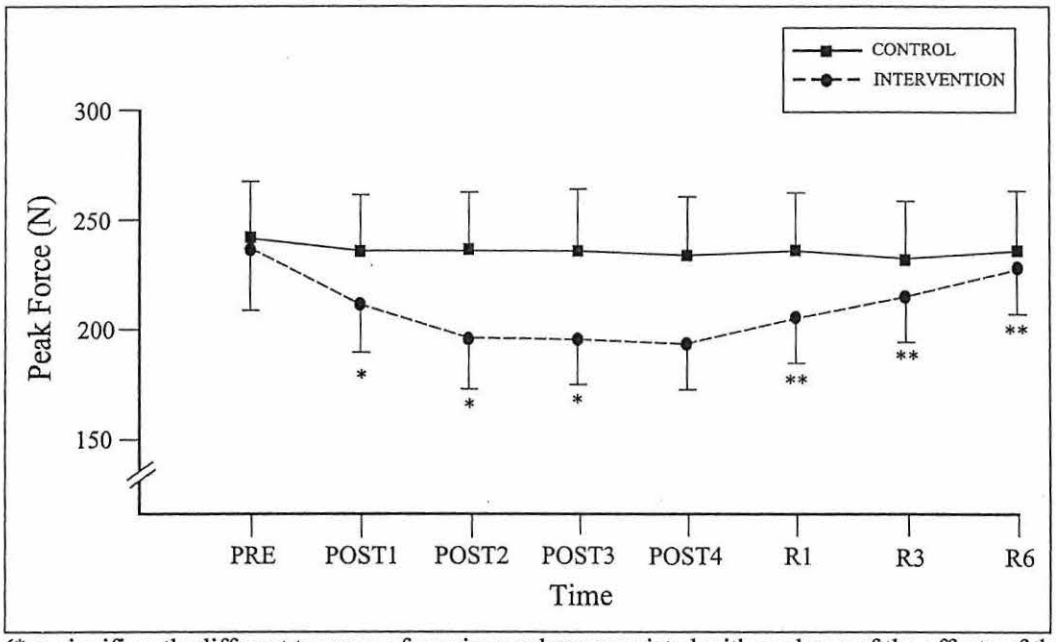
7.4 Results

7.4.1 *Volitional muscle activation*

Indices of volitional peak force (PF_V)

Repeated measures ANOVA revealed a significant condition (control; intervention) by time (pre; post1; post2; post3; post4) interaction ($F_{[2.4, 44.8\ GG]} = 3.2$, $p < 0.05$) which showed that while absolute strength was preserved over the control period, the exercise intervention induced a significant reduction in absolute PF_V performance. *A priori* tests comparing the relative differences in the control and intervention conditions showed that comparisons of the first trial (pre) vs. the second trial (post1), the mean of the first two trials vs. the third (post2) and the mean of the first three trials vs. the fourth (post3) contributed most to this effect ($F_{[1, 19]} = 5.9, 14.9, 6.1$, respectively, $p < 0.05$). This implies a progressive increase in fatigue up to the cessation of the third bout of exercise (post3). The actual losses in PF_V performance associated with post1, post2 and post3 were 10.3%, 15.9%, 15.8%, respectively by comparison to pre-exercise levels. Repeated measures ANOVA of recovery measures revealed a significant condition by time interaction ($F_{[3, 57]} = 17.1$, $p < 0.001$) which showed that while absolute strength was preserved over the control period, absolute PF_V performance increased to beyond baseline values (post 4). *A priori* tests comparing the relative differences in performance in the control and intervention conditions showed that all comparisons contributed significantly to this effect ($F_{[1, 19]} = 4.5, 28.7, 17.1$, respectively, $p < 0.05$). This implies recovery of absolute PF_V performance was evident at 1 minute following cessation of exercise and continued to 6 minutes following exercise at which point it reached 96.6% of pre-exercise values (please see figure 7.2 and table 7.1).

Figure 7.2. The effects of four episodes of maximal static exercise and subsequent recovery on the volitional peak force performance (PF_V) of the knee flexors (group mean ± SD).



(* = significantly different to mean of previous values associated with analyses of the effects of the exercise intervention [pre to post4]; ** = significantly different to mean of previous values associated with analyses of recovery [post4 to R6]).

Indices of volitional electromechanical delay (EMD_V)

Repeated measures ANOVA revealed a significant condition by time interaction ($F_{[4, 76]} = 2.7$, $p < 0.05$) which showed that while performance was preserved over the control period, the exercise intervention induced a significant reduction in absolute EMD_V. *A priori* tests comparing the relative differences in the control and intervention conditions showed that the comparison of the first trial (pre) vs. the second trial (post1) contributed most to this effect ($F_{[1, 19]} = 6.5$, $p < 0.05$). This implies the significant 25.5% increase in EMD_V following the first exercise bout by comparison to pre-exercise values (55.3 ± 11.8 ms vs. 69.4 ± 24.5 ms, respectively) was maintained up to post4. Repeated measures ANOVA of recovery values revealed a main effect for condition only ($F_{[1, 19]} = 10.0$, $p < 0.05$), showing absolute values during the control period to be significantly less than during the intervention

condition. This suggests that the impairment of EMD_V performance associated with the exercise intervention was sustained throughout the 6 minutes following the cessation of exercise (please see figure 7.4).

Indices of volitional rate of force development (RFD_V)

Repeated measures ANOVA revealed main effects for time ($F_{[2.6, 49.4\ GG]} = 3.19$, $p < 0.05$) and condition ($F_{[1.19]} = 24.9$, $p < 0.001$), showing absolute values during pre and post1 to be superior compared to post2, post3 and post4 and control values to be significantly greater than during the intervention condition. Repeated measures ANOVA of recovery scores showed a main effect for condition only ($F_{[1, 19]} = 14.0$, $p < 0.05$), indicating values associated with the control condition were significantly greater than by comparison to the intervention. While some performance decrements resulting from the exercise intervention may have been experienced by some individuals within the group, the heterogeneity of performance scores of the sample population, indicated by the large group standard deviations (please see table 7.1), may have contributed to a lack of experimental design sensitivity and partly explain the failure to challenge effectively the null hypothesis of no interaction associated with these analyses.

Indices of volitional time to half peak force ($T_{½V}$)

A significant main effect for condition associated with the repeated measures ANOVA showed that $T_{½V}$ performance was significantly superior during the control task by comparison to the intervention ($F_{[1, 19]} = 5.7$, $p < 0.05$). Repeated measures ANOVA of recovery values revealed no significant main effects or interactions.

Table 7.1. Group mean scores of indices of volitional neuromuscular performance associated with the intervention condition (mean ± SD).

Index		Time							
		Pre 1	Post 1	Post 2	Post 3	Post 4	R1	R3	R6
PF_V	(N)	235.3 ± 54.7	211.1 ± 46.6	198.0 ± 44.8	198.1 ± 38.4	194.9 ± 38.9	206.3 ± 39.9	221.2 ± 42.5	227.4 ± 44.5
EMD_V	(ms)	55.3 ± 11.8	69.4 ± 24.5	68.1 ± 23.5	66.5 ± 26.9	67.4 ± 22.0	66.6 ± 19.1	65.0 ± 17.3	64.4 ± 16.9
RFD_V	($N.s^{-1}$)	1649 ± 850	1339 ± 521	1130 ± 523	1151 ± 414	1106 ± 485	1099 ± 603	1151 ± 561	1269 ± 554
$T_{½V}$	(ms)	114.0 ± 40.2	106.4 ± 45.8	112.3 ± 48.0	106.1 ± 29.0	105.2 ± 25.7	119.9 ± 40.9	114.7 ± 38.7	113.6 ± 37.8

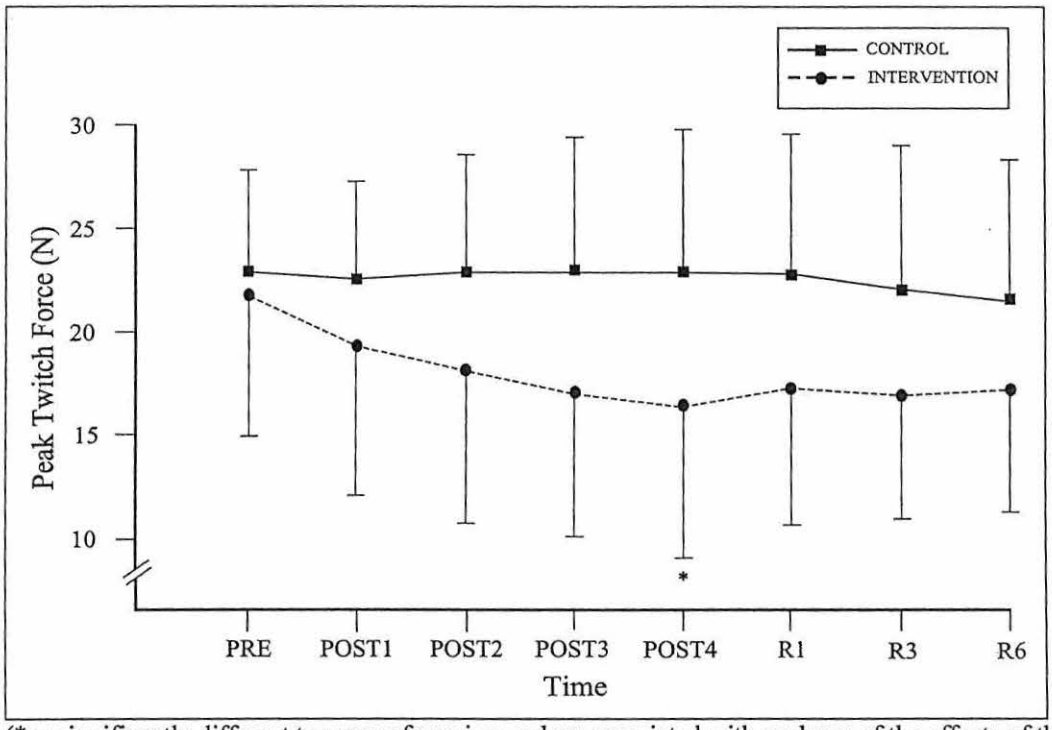
7.4.2 *Magnetically evoked muscle activation*

Indices of magnetically evoked peak twitch force (P_TF_E)

Repeated measures ANOVA revealed a significant condition by time interaction ($F_{[2.3, 44.5\ GG]} = 3.2$, $p < 0.05$) which showed that while performance was preserved over the control period, the exercise intervention induced a significant reduction in absolute P_TF_E. *A priori* tests comparing the relative differences in the control and intervention conditions showed that the comparison of the mean of the first four trials (pre, post1, post2, post3) vs. the fifth trial (post4) contributed most to this effect ($F_{[1, 19]} = 6.4$, $p < 0.05$). This implies P_TF_E performance was significantly impaired following the fourth exercise bout (23.3% decrease compared to pre-exercise values) (please see figure 7.3). Repeated measures ANOVA of recovery scores revealed a significant main effect for condition only ($F_{[1,19]} = 20.2$, $p < 0.001$), showing values during the control period to be significantly greater than during the intervention condition. This suggests that the impairment of P_TF_E

performance associated with the last exercise bout was sustained throughout the 6 minutes following the cessation of the exercise.

Figure 7.3. The effects of four episodes of maximal static exercise and subsequent recovery on the magnetically evoked peak twitch force performance (P_TF_E) of the knee flexors (group mean ± SD).



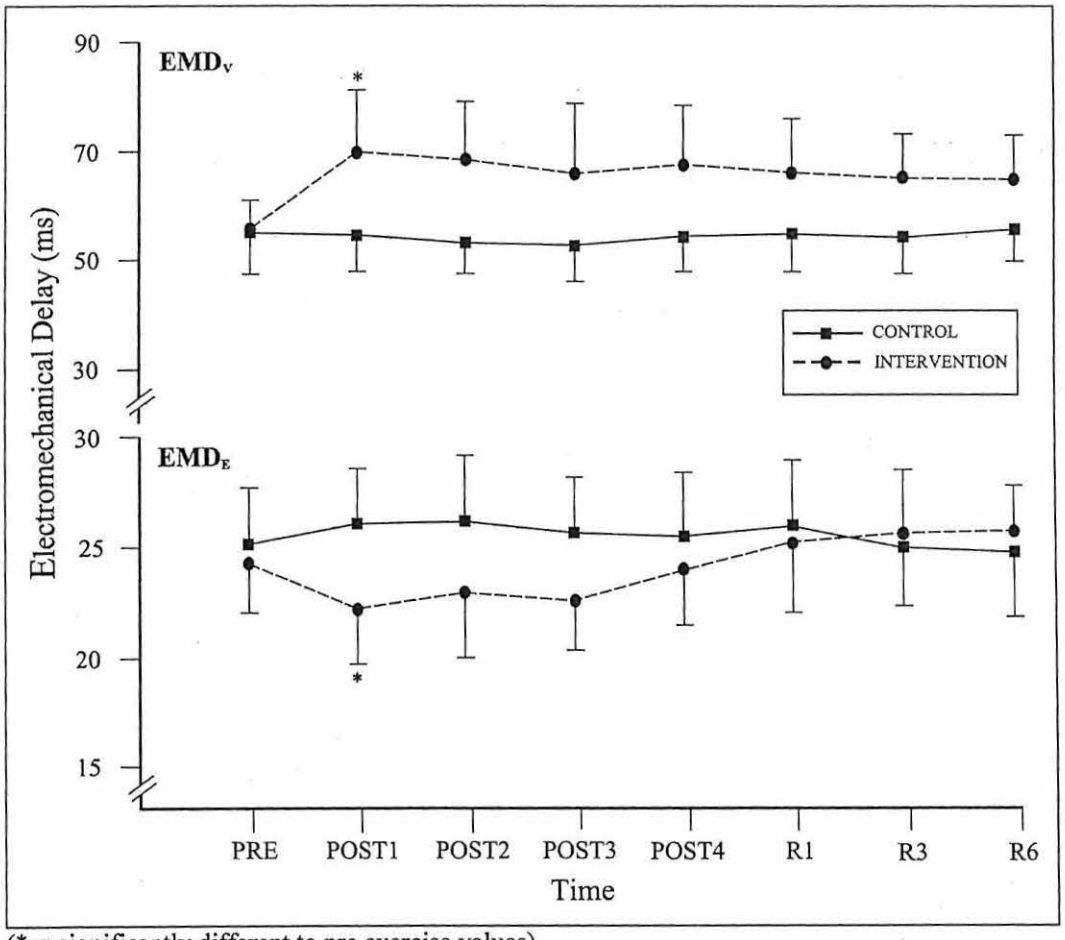
(* = significantly different to mean of previous values associated with analyses of the effects of the exercise intervention [pre to post4]).

Indices of magnetically evoked electromechanical delay (EMD_E)

A significant condition by time interaction associated with the repeated measures ANOVA showed that while absolute EMD_E performance was preserved during the control task, the exercise task elicited a decrease in EMD_E scores ($F_{[2.6, 49.6\ GG]} = 3.18$, $p < 0.05$). *A priori* tests comparing the relative differences in the control and intervention conditions showed that comparison of the first trial (pre) vs. the second trial (post1) contributed most to this effect ($F_{[1, 19]} = 5.9$, $p < 0.05$). This suggests the exercise intervention was associated with a 10.0% improvement in EMD_E performance following the first exercise bout by comparison to pre-exercise values

(24.5 ± 4.7 ms vs. 22.1 ± 5.0 ms), which was maintained up to assessment point post4. Repeated measures ANOVA of recovery scores revealed no significant main or interaction effects. This implies that EMD_E performance scores were similar between treatment conditions throughout the recovery period.

Figure 7.4. The effects of four bouts of maximal static exercise and subsequent recovery on the volitional and magnetically evoked electromechanical delay performance (EMD_V, EMD_E, respectively) of the knee flexors (group mean ± SD).



(* = significantly different to pre exercise values).

Indices of magnetically evoked rate of force development (RFD_E)

Repeated measures ANOVAs revealed no significant main or interaction effects over the exercise or recovery periods.

Indices of magnetically evoked time to half peak twitch force ($T_{½E}$)

Repeated measures ANOVA revealed a significant condition by time interaction ($F_{[2.8, 53.1\ GG]} = 3.18$, $p < 0.05$) . *A priori* tests comparing the relative differences in the control and intervention conditions of $T_{½E}$ performance showed that the comparisons of the mean of the first two trials (pre, post1) vs. the third trial (post2) and the mean of the first four trials vs. the fifth trial (post4) contributed most to this effect ($F_{[1, 19]} = 6.9, 11.1$, respectively $p < 0.05$). This implies a progressive improvement of $T_{½E}$ performance of 8.2% and 9.1% compared pre-exercise values at assessment points post2 and post4, respectively. Repeated measures ANOVA of recovery values shows a main effect for condition only ($F_{[1, 19]} = 11.8$, $p < 0.05$), this implies that the potentiation of $T_{½E}$ performance was maintained throughout the 6 minutes following the cessation of the intervention (please see table 7.2).

Table 7.2. Group mean scores of indices of magnetically evoked neuromuscular performance associated with the intervention condition (mean ± SD).

Index		Time							
		Pre 1	Post 1	Post 2	Post 3	Post 4	R1	R3	R6
P_TF_E	(N)	21.5 ± 13.6	19.1 ± 14.5	18.3 ± 14.8	17.0 ± 13.6	16.4 ± 14.7	17.3 ± 13.2	16.9 ± 11.8	17.3 ± 12.0
EMD_E	(ms)	24.5 ± 4.7	22.1 ± 5.0	23.5 ± 5.9	22.6 ± 4.5	24.2 ± 5.4	25.4 ± 7.0	25.6 ± 7.0	25.8 ± 7.0
RFD_E	($N.s^{-1}$)	1074 ± 649	975 ± 787	986 ± 806	965 ± 717	962 ± 816	1017 ± 728	981 ± 660	986 ± 642
$T_{½E}$	(ms)	20.8 ± 5.9	20.8 ± 5.1	19.1 ± 3.4	19.4 ± 3.9	18.9 ± 3.5	18.8 ± 3.2	18.1 ± 3.8	18.0 ± 3.2

7.5 Discussion

7.5.1 *Volitional neuromuscular performance*

The results show the exercise intervention induced fatigue in the knee flexors, characterised by a significant decrease in PF_V which was progressive from pre- to immediately following the third bout of exercise (10.3% at post1 to 15.8% at post3) (please see figure 7.2). The magnitude of PF_V performance decrement observed in the current study is congruent with the extent of performance loss associated with match play in team games such as soccer (Gleeson et al., 1998b), and is similar to previous reports of the knee extensors following six bouts of thirty seconds maximal static muscle activation (15.2% decrease) at a similar joint angle (Chan et al., 2001). In addition, following the first episode of maximal exercise, the observed impairments to PF_V and EMD_V performance of 10.3% and 25.5%, respectively in the current study are comparable to the performance losses experienced by females in the previous study (chapter 6.0) subsequent to a similar episode of exercise (10% and 19.3%, respectively). These results suggest a similarity in 'dose-response' characteristics associated with the initial fatigue task for the two populations in these investigations.

There was a progressive decrease in PF_V performance subsequent to the repeated episodes of exercise. However, the impairment of EMD_V capabilities following the first bout (25.5%) was largely sustained at this level across all subsequent episodes of fatiguing exercise (please see figure 7.4). Given that the majority of EMD is determined by the time to stretch the SEC (Cavanagh and Komi, 1979; Norman and Komi, 1979), increases in compliance of the SEC may have been instrumental in this change (Zhou et al., 1998). Yet, due to the static testing conditions, further

attenuation of EMD_V performance may have been ameliorated by competing influences such as exercise-related hyperaemia that would tend to increase musculoskeletal stiffness (McComas, 1996) and stress relaxation in which connective tissue and muscle-tendon units subjected to a constant stress, elongate and become more compliant mechanically over time (Stone, 1992). The relative importance of such factors may be limited during some conditions of dynamic exercise, given that greater impairments to EMD have been reported by Zhou et al. (1996) following four bouts of thirty seconds maximal sprint cycling (67% increase in EMD compared to pre-exercise values). As alluded to previously, impairments to the EMDv of the knee flexors may be commensurate with a reduction to the temporal capability to harness dynamic joint forces and the avoidance of knee injury (Mercer et al., 1998). Since recovery of EMD_V performance capabilities had not occurred within six minutes following the exercise protocol, serial episodes of acute maximal activity may be associated with a prolonged increased risk of knee ligamentous injury during continued sports participation. This may be of concern to the female athlete who's specific anatomical and bio-physiological capacities may already predispose them to increased risk of ACL injury compared to male counterparts (Hutchinson and Ireland, 1995; Wojtys et al., 1998; Harmon and Ireland, 2000), particularly those who participate in team games characterised by intense unpredictable mechanical loading of the knee (McInnes et al., 1995). Recovery of PF_V performance, however, was rapid, reaching 87.7% of pre-exercise values by 1 minute following cessation of the exercise intervention and continued to increase to 96.6% of pre-exercise levels by 6 minutes (please see figure 7.1 and table 7.2). This rapid phase of recovery of PF_V is similar to previous research (Bilcheck et al., 1990; Sahlin and Seger, 1995; Zhou, 1996) and is consistent with

the time course of phosphocreatine resynthesis (Sahlin and Ren, 1989). The differential changes in estimates of PF_V and EMD_V performance throughout exercise and acute recovery may suggest measures of these indices are representative of different capacities within the neuromuscular system. Subsequent computation of correlations between these indices of performance ($r = 0.33 \pm 0.29$, $p < 0.05$ (group mean ± SD)) may substantiate that factors that primarily affect the capabilities to produce peak force, such as Ca^{2+} release and the speed of cross bridge cycling (Metzger and Fitts, 1987; Fitts, 1996; Kent-Braun, 1999; Yeung et al., 1999), have only limited influence on EMDv performance. As mentioned previously, it is generally accepted that the majority of the EMD comprises the time to stretch of the SEC (Cavanagh and Komi, 1979; Norman and Komi, 1979) and as such, factors that may induce alterations in this elastic component, including exercise-induced changes in muscle temperature (Zhou et al., 1998), may be the principle limiting factor to EMDv performance capabilities. Accordingly, compliance of the SEC following serial episodes of exercise may be associated with a longer time course for recovery compared to the restoration of elements limiting force production.

Despite the exercise protocol causing cumulative fatigue, no significant changes to the temporal force parameters $T_{½V}$ and RFD_V were observed, contrary to the previous investigation (6.0). However, reduced levels of absolute peak force and large intra-population coefficients of variation of approximately 51% and 42% associated with some control and intervention group mean scores for RFD_V and $T_{½V}$, respectively, in addition to an associated reduction in experimental design sensitivity in general, may account for these findings. As such, any subtle decreases

in RFD_V performance experienced by some individuals, for example, could have been rendered undetectable.

7.5.2 *Implications for the female sports performer*

Given the impairment to PF_V capabilities and the focus of current research linking EMD to knee injury (e.g. Gleeson et al., 1997; Gleeson et al., 1998a; Mercer et al., 1998; Gleeson et al., 2000), consecutive episodes of maximal exercise may be sufficient to substantively affect the knee injury avoidance capabilities of the female athlete. This fatigue-related loss of neuromuscular performance may persist for several minutes following the cessation of exercise and may render the athlete even more vulnerable to injury should they decide to reinitiate high-intensity exercise involving the knee flexors within this period of time.

7.5.3 *Magnetically evoked neuromuscular performance*

Magnetic stimulation, which activates the fastest motor units (Maertens de Noodhout, 1991), provides an estimate of the true performance capacity of the neuromuscular system (King and Chippa, 1989). The current exercise protocol, which induced a significant decrease in P_TF_E (23% decrease, evident at assessment point post4 until the last assessment occasion (R6)), may suggest that such high intensity exercise can be associated with a reduction in the ultimate force generating capacity of the neuromuscular system. This may be due to a need to recruit more heavily from the capacity of large, fast motor units to preserve as much as possible the volitional neuromuscular capabilities during repeated episodes of maximal exercise. Reductions in peak twitch force of the knee extensors have been reported previously following serial episodes of maximal exercise (56% decrease) (Zhou,

1996) and prolonged sub-maximal exercise (45% decrease) (Polkey et al., 1996), subsequent to electrically and magnetically evoked activation of muscle, respectively. The decrease in P_TF_E up to R6 in the current study may highlight the potential potency of acute periods of match-play in team games and a prolonged impairment of the capacity to avoid injury. However, despite the exercise-related impairments to indices of volitional (PF_V, EMD_V) and magnetically evoked (P_TF_E) neuromuscular performance, the temporal capacity to produce muscle force was improved.

The potentiation of EMD_E and $T_{½E}$ performance capacity following the first episode of exercise of 10.0% and 8.2%, respectively is similar to the results of the previous study (chapter 6.0) (21.3% decrease in EMD_E). Although the serial episodes of exercise caused cumulative fatigue up to assessment point post3 (please see figure 7.2). Analyses revealed that, the improved temporal performance capacity remained until the end of the exercise protocol (please see figure 7.4) and actually improved further for $T_{½E}$ at post3 (further 1% decrease). These processes, which may reflect adaptations to conductive (Zhou et al., 1998), contractile (O'Leary et al., 1997; Rassier and MacIntosh, 2000) or elastic factors (Stone, 1992; McComas, 1996), may be commensurate with the potential to compensate for fatigue-related impairments. The current results together with the findings of the previous study (chapter 5.0) offers accumulating evidence to suggest that acute muscle fatigue of the dynamic joint stabilisers might enable temporal improvements that may compensate for reduced absolute force generating capabilities. In scenarios involving potentially harmful joint perturbations, the timely production of meaningful levels of muscle force may be more important than the absolute strength

capabilities of the musculature to prevent knee ligamentous injury. The present results also suggest that such potentiation of the temporal capacity of the knee flexors may remain throughout the ensuing period of recovery whereas volitional neuromuscular performance capabilities and P_TF_E are still impaired. In order to effectively compensate for the impairment of the voluntary neuromuscular performance capabilities (PF_V, EMD_V), however, the protective inhibitory mechanisms that restrict volitional access to the full capacity of large high threshold motor units (Tsuji and Nakamura, 1988; Zhou et al., 1995; Hopkins and Ingersoll, 2000; Gleeson, 2001) must be down-regulated. Presently, evidence of this appears to be limited, demonstrated by the consistently longer latencies associated with EMD_V (e.g. 69.4 ms) by comparison to EMD_E (e.g. 22.1 ms) throughout the intervention and control periods (please see figure 7.4 and tables 7.1 and 7.2), though a reduction in estimates of P_TF_E following the exercise intervention may suggest this is possible. True emergency scenarios may indeed be associated with the capability to overcome potential neuromuscular inhibitory mechanisms, however, ethical and kinanthropometric considerations dictate that such assessment scenarios are very difficult to safely create. As such, the current estimates of performance, obtained in a laboratory setting, may represent an underestimation of the neuromuscular capabilities available to an individual during real threats to knee joint integrity, despite the best efforts of the participants to maximally activate their musculature.

As alluded to previously, different mechanisms may mediate peak force and EMD capabilities, which may also account for the differential changes in performance under magnetically evoked conditions. However, despite impairment of P_TF_E

suggesting some change to the capacity of the neuromuscular system to produce force, the improvement to EMD_E could be the most influential change to performance, since the capacity to rapidly initiate muscle force may a principle factor determining the level of joint protection (Gleeson et al., 1998; Gleeson et al., 2000; Mercer et al., 1998). This acute adaptation may, therefore, represent a vital neuromuscular compensatory mechanism to counteract possible prolonged impairments to the temporal voluntary performance capabilities which may place the female athlete at increased risk of knee injury by comparison to un-fatigued conditions.

7.5.4 *Summary*

The serial exercise task induced cumulative fatigue in the knee flexors of the present sub-sample of female team games players of up to 15.9% (PF_V), which recovered to 96.6% of pre-exercise values by six minutes following cessation of the protocol. An associated 25.5% increase in EMD_V following the first episode of exercise was maintained throughout the remainder of the intervention and recovery. Such impairment to EMD_V, of the knee flexors may place the female athlete at increased and prolonged risk of knee injury, particularly in the presence of reduced PF_V capabilities. Magnetically evoked responses confirmed the findings of the previous study (chapter 6.0), whereby the temporal indices EMD_E and $T_{½E}$ were potentiated throughout the period of impairment to volitional neuromuscular capabilities. Potentiation of the temporal capacity of the knee flexors may be a vital strategy to enable joint protection during periods of decreased volitional neuromuscular performance capabilities. The utility of this type of physiological

potentiation process, however, is reliant on the timely recognition of potential noxious stimuli and the down-regulation of routine protective inhibitory processes restricting access to the total neuromuscular capacity (Hopkins and Ingersoll, 2000; Gleeson, 2001). It is plausible that in some circumstances, an episode of fatiguing exercise may actually facilitate rather than impede neuromuscular performance in the knee flexors and ultimately enhance the capability of the joint to resist injury.

Interventions:

Chapter 8

EFFECTS OF EXERCISE-INDUCED MUSCLE DAMAGE ON AGONIST AND ANTAGONIST FATIGUE-RELATED VOLITIONAL AND MAGNETICALLY EVOKED NEUROMUSCULAR PERFORMANCE

8.1 Abstract

The purpose of this study was to examine the effects eccentric exercise-induced muscle damage and a static fatigue task in the knee flexors on the voluntary and magnetically evoked neuromuscular performance of the agonist and antagonist muscle groups in males. Voluntary and magnetically evoked indices of neuromuscular performance of the knee flexors (agonist) of the dominant leg of seven males (age: 28.3 ± 7.02 yrs; height 1.84 ± 0.05 m; body mass 83.0 ± 13.9 kg [mean ± SD]) were obtained prior to, at pre-determined periods following three treatment conditions. The treatment conditions consisted of (i) an eccentric exercise-induce muscle damage condition (EIMD) of the knee flexors of the preferred leg (performed on the first assessment occasion), in addition, to a static fatiguing exercise task of the same muscle group, performed on every assessment occasion; (ii) a control condition (CON2) of equivalent duration to the intervention, consisting of the static fatiguing exercise task only; (iii) and a further control condition (CON1) of equivalent duration to the EIMD and static fatiguing exercise interventions, consisting of no exercise. Performance measures were obtained prior to (pre) and up to 168 hours (168h) following each treatment condition, in addition to prior to and immediately following the static exercise task (or equivalent period of rest). Assessment of volitional and magnetically evoked performance of the knee extensors of the preferred leg was also obtained at the start of each assessment occasion. The results strongly suggest that the eccentric exercise induced muscle damage in the knee flexors, characterised by an increase in serum CK values ($p < 0.001$), peaking at 72h and ratings of perceived soreness ($p < 0.001$), consistent with the DOMS phenomenon, commencing at 24h and peaking at 48h. The results also

showed that EIMD was associated with impaired volitional peak force (PF_V) (up to 37.5%, $p < 0.001$) and rate of force development (RFD_V) (up to 65.2%, $p < 0.01$) performance of the knee flexors and a preserved electromechanical delay (EMD_V). Prior muscle damage did not exacerbate the magnitude of the fatigue-related changes associated with the static exercise task compared to CON2, however, further reductions to contractile capabilities in addition to the effects of muscle damage were observed subsequent to fatigue. The results of the antagonist muscle group (knee extensors) analyses indicate superior absolute neuromuscular performance levels compared to the agonist muscle group (knee flexors). The effects of EIMD on the antagonist was limited to a minor decrease in RFD_V capabilities of up to 18.0% (at 48h) compared to pre-EIMD levels. Such changes to the maximal force generating capabilities of the active knee stabilisers may present substantive challenges to the neuromuscular system to protect the ACL during mechanical loading of the knee joint, especially at knee angles proximal to full extension. The performance capacity of the neuromuscular system of the knee flexors, as measured by magnetic stimulation, was maintained during EIMD and even potentiated, in the case of EMD_E, ($p < 0.01$) following fatigue. The preservation of such neuromuscular performance capacity may be evidence of a vital compensatory strategy to help prevent the sports performer becoming injured on each occasion when they experience fatigue and/or symptoms of exercise-induced muscle damage. However, this is entirely dependent on whether or not potential inhibitory mechanisms that may restrict volitional access to the full capacity of motor units within a given muscle (Hopkins and Ingersoll, 2000) can be down-regulated during critical periods of mechanical joint loading.

8.2 Introduction

The current series of investigations (chapters 6.0, 7.0) has confirmed that static maximal intensity fatiguing exercise can cause impairment to various aspects of volitional neuromuscular capabilities of the knee flexors. The extent of performance loss appeared to be consistent with the findings from previous research involving the knee flexors (Gleeson et al, 1997; Gleeson et al., 1998a; Mercer et al., 1998) and other muscle groups (Zhou et al., 1996; Yeung et al., 1999; Chan et al., 2001). In addition, it has been shown that these decrements to performance may persist following cessation of exercise. The possible consequences of such changes have been explored in relation to knee joint stability of the sports performer. However, research findings from the estimation of maximum neuromuscular performance capability by means of electromagnetic stimulation of peripheral nerves has shown that the neuromuscular system may have the potential to utilise certain compensatory mechanisms that may counteract the effects of such fatigue-induced impairments. The results have shown that indices of evoked neuromuscular performance such as EMD_E, are at least preserved and in some cases enhanced, during maximal volitional muscle fatigue. Therefore, it would seem plausible that at any moment in time, a proportion of the pool of motor units available for volitional recruitment are routinely kept in 'reserve' for those occasions when true emergency threats have been perceived. Furthermore, since evoked indices of temporal performance are consistently superior to their volitional counterparts, it seems likely that such motor units will be those associated with high activation thresholds and superior capability for velocity of shortening and tension when activated. A corollary of this interpretation is that assessments of

neuromuscular performance by means of magnetic stimulation may offer greater insights into the performance capability that might be available to the sports performer in emergencies situations where there is a critical level of threat to the stability of the joint system.

Following unaccustomed high intensity exercise, skeletal muscle is susceptible to ultrastructural damage (Armstrong et al., 1983; Jones et al., 1989; Byrne and Eston, 1998). This type of exercise usually features a large eccentric component, whereby the muscle is lengthened while developing force (Latash, 1998). While the possibility of selective recruitment of fast-twitch motor units under eccentric conditions remains uncertain (Enoka and Fuglevand, 2001), it appears that fast fibres are more susceptible to damage (Friden and Lieber, 1992; Linnamo et al., 2000; Brockett et al., 2002). Accordingly, the protective capability of the neuromuscular system may also be compromised. Delayed onset of muscle soreness (DOMS) is often associated with high intensity eccentric exercise and may be experience by the sports performer who recommences training, match play or even rehabilitation following a prolonged period of inactivity or injury. Although the effects of muscle damage on the susceptibility to further injury remains relatively unknown, the recent findings of an audit of injuries of all English professional soccer clubs may suggest that an increased risk exists. It was observed that approximately 13% of all injuries sustained over two competitive seasons (1997/1998 – 1999/1999) occurred during pre-season training (Hawkins et al., 2001), prior to which professional players are afforded a period of rest of at least two full months. Given that physical conditioning can attenuate the symptoms of muscle damage (McHugh et al., 1999), potential 'detraining' throughout this eight-

week period may render players more susceptible to muscle damage and perhaps to further injury.

Exercise-induced muscle damage is believed to be caused by excessive stress on a small number of active muscle fibres during eccentric activation compared to concentric and isometric activation (McHugh et al., 2000). A major functional consequence of which is an immediate and prolonged loss of muscle force (Byrne et al., 2001; Nokasa et al., 2001). For example, reductions in peak force of up to 69% have been reported following eccentric exercise (Rinard et al., 2000), which often requires several days to fully recover (Rinard et al., 2000; Nokasa et al., 2001). In addition, substantive impairments to rate of force development (38% decrease) have also been observed following sub-maximal stretch-shortening exercise (Strojnik and Komi, 2000). Despite the focus of current literature highlighting the potential importance of EMD within a conceptual model for knee joint stability (Gleeson et al., 1997; Gleeson et al., 1998a; 1998b; Mercer et al., 1998; Gleeson et al., 2000), there has been limited investigation in to the temporal capability of the neuromuscular system to initiate muscle force following eccentric exercise. While it seems that muscle damage induced by means of electrically evoked eccentric muscle actions may prolong the associated neuromuscular delay (Brown et al., 1996), volitional sub-maximal stretch-shortening exercise has not been associated with impairment to electrically evoked EMD despite considerable decrements to volitional peak force and rate of force development capabilities (Strojnik and Komi, 2000). Methods of evoked muscle activation may recruit a greater proportion of fast-twitch motor units compared to volitional actions (King and Chippa, 1989) and may, therefore, have the potential for greater levels of muscle

damage. Accordingly, possible impairments to EMD may be magnified following electrically induced muscle damage. Absence of impairments to electrically evoked EMD, yet the presence of substantive decrements to force generating capabilities as observed by Strojnik and Komi (2000) may be explained by potential modifications to the properties of the SEC, induced by possible eccentric exercise-induced muscle length adaptations (Morgan and Allen, 1999). Clearly, further investigation is required to elucidate the effects of exercise-induced muscle damage on the temporal capability of the dynamic knee stabilisers to initiate force. Furthermore, if mechanical damage to the muscle is biased towards fast-twitch muscle fibres (Linnamo et al., 2000), the pool of motor units most equipped to rapidly initiate muscle force and protect joint integrity may be compromised. It is plausible that the effects of eccentric exercise-induced muscle damage on magnetically evoked assessment of performance may reveal a reduced capacity for neuromuscular performance. Such an observation would tend to mediate against the routine availability to the performer of potential compensatory mechanisms in an emergency subsequent to this type of stressful exercise.

The pain (DOMS) (Jones et al., 1989; Brown et al., 1997) and possible intramuscular swelling (Armstrong et al., 1983; Foley et al., 1999) responses following eccentric exercise may also effect changes to the central drive to the involved (Morgan and Allen, 1999) and reciprocal muscle groups (Leger and Milner, 2001). Although some research involving single muscle preparations disputes central drive modifications as a major factor influencing performance of the involved musculature subsequent to exercise-induced muscle damage (Ingalls et al., 1998), a more recent investigation demonstrating losses to proprioceptive

capabilities, as measured by angle and force replication tasks, may imply some changes to central activity (Saxton et al., 1995). Modifications to afferent activity of a muscle symptomatic of exercise-induced muscle damage by the aforementioned mechanisms, coupled with possible disruption to muscle spindles, may be associated with changes in the reciprocal inhibition of the antagonist musculature. For example, Leger and Milner (2001) reported increased antagonist activity of the wrist flexors during sustained activation of the wrist extensors following muscle damage. Such changes to reciprocal muscle group activity in addition to the involved musculature may have further implications for the dynamic stabilisation for the knee joint and potential injury avoidance capabilities.

The literature examining the effects of fatigue clearly presents evidence to show that acute maximal muscle activation tasks can induce impairment to the neuromuscular performance capabilities (Horita and Ishiko, 1987; Zhou et al., 1996; Gleeson et al., 1997; Yeung et al., 1999). In addition, high intensity unaccustomed exercise is also associated with substantive and prolonged impairment to the force generating capability of the involved musculature (Jones et al., 1989; Byrne et al., 2001). Currently, however, there is limited investigation to the concomitant effects of acute maximal exercise in muscle symptomatic of damage. There is some suggestion that exercise-induced muscle damage may cause premature fatigue compared to healthy muscle as measured by lactate accumulation during sub-maximal incremental exercise (Gleeson et al., 1995). Further study of changes to neuromuscular performance may provide a better understanding of the capabilities of the dynamic muscle stabilisers under potentially high stress exercise conditions

and enable the implementation of appropriate prophylactic strategies to help reduce the risk of knee injury.

The aim of this study is to examine the effects eccentric exercise-induced muscle damage and a static fatigue task in the knee flexors on the on the voluntary and magnetically evoked neuromuscular performance of the agonist and antagonist muscle groups in males.

8.3 Methods

8.3.1 *Participants*

Seven males (age: 28.3 ± 7.02 yrs; height 1.84 ± 0.05 m; body mass 83.0 ± 13.9 kg [mean ± SD]) gave their informed consent to participate in this study. All participants were regularly involved in exercise (at least 3 times per week) and were asymptomatic at the time of assessment. Participants had not engaged in any resistance training of the involved musculature for six months prior to assessment, this was necessary to avoid the potential attenuation of the muscle damage response (Byrne and Eston, 1998). Participants were also instructed to refrain from strenuous physical activity for the twenty-four hours prior to the test and maintain constant exercise levels throughout the experimental period. Individuals were requested not to ingest any pain-relieving medication in order to prevent the confounding effect on the perception of muscle soreness. The latter was used as an indirect indicator of muscle damage in this study. The assessment protocols were approved by the University of Wales, Bangor, Human Performance Ethics Review Committee. The time commitment of each participant within this study approximated 20 hours.

While it has been acknowledged throughout this document that females are at increased risk of ACL injury compared to male counterparts, the current sample population was comprised of males for two reasons. Firstly, little is known about the temporal neuromuscular responses to muscle damage. As such, the sample population and the method utilised to induce muscle damage were chosen to be commensurate with those used in previous research within this laboratory to maximise the efficacy of the experimental intervention. Secondly, participants were representative of a convenience sample of individuals who were willing and able to

comply fully with the protocol of the study despite the necessity of a substantial time-commitment for each participant (> 20 hours).

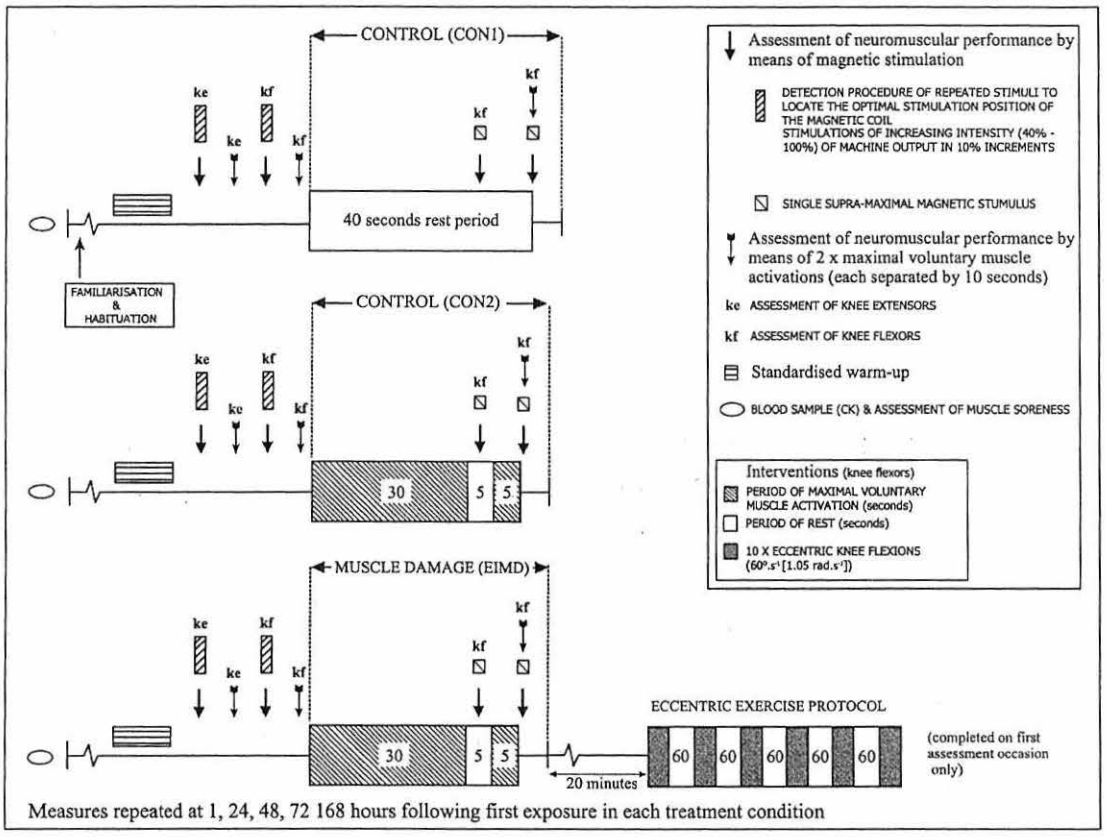
8.3.2 *Experimental procedures*

Following habituation procedures, participants completed a standardised warm-up consisting of five minutes cycle ergometry (90 Watts) and a further five minutes of static stretching of the involved musculature prior to testing. Participants were then secured in the appropriate position on a custom-built dynamometer (modified from Gleeson et al., 1995).

Assessments of volitional and magnetically evoked neuromuscular performance of the knee flexors of the preferred leg were obtained prior to, at pre-determined periods following three treatment conditions, in accordance with the protocols and experimental conditions outlined in chapter 3.0 (sub-chapter 3.2), please see figure 3.1 for participant and dynamometer orientation (p 74). Each of the treatment conditions were separated by at least one week and consisted of (i) an exercise-induce muscle damage condition (EIMD) which required participants to perform an eccentric-exercise trial of 6 x 10 maximal isokinetic muscle activations of the knee flexors of the preferred leg (on the first assessment occasion). In addition, a static fatiguing exercise task of 35 seconds MVMA of the same muscle group was performed on every assessment occasion; (ii) a control condition (CON2) of equivalent duration to the intervention, consisting of only the static fatiguing exercise task; (iii) and a further control condition (CON1) of equivalent duration to the EIMD and static fatiguing exercise interventions, consisting of no exercise. Performance measures were obtained prior to (pre) and at 1 hour (1h), 24 hours

(24h), 48 hours (48h), 72 hours (72h) and 168 hours (168h) following each treatment condition. Within each assessment occasion, measures of volitional and magnetically evoked performance of the knee flexors of the preferred leg were also taken prior to and immediately following the static fatiguing exercise task (or equivalent period of rest) (please see figure 8.1). The CON1 and CON2 treatment conditions were presented in random order to offset any sequencing effects. However the EIMD condition was performed last due to the extended period of recovery required to ensure total restoration of muscle function following eccentric exercise induced damage (Brown et al., 1997). Assessment of volitional and magnetically evoked performance of the knee extensors of the preferred leg was also obtained at the start of each assessment occasion (please see figure 8.1) in accordance with the protocols and experimental conditions outlined in chapter 3.0 (sub-chapter 3.3), please see figure 3.2 for participant and dynamometer orientation (p 77).

Figure 8.1. Schematic of the protocol to assess the effects of exercise-induced muscle damage on agonist and antagonist fatigue-related volitional and magnetically evoked neuromuscular performance.



8.3.3 *Indices of volitional neuromuscular performance*

The estimates of volitional neuromuscular performance of the knee flexors of the preferred leg were static peak force (PF_V), electromechanical delay (EMD_V) and rate of force development (RFD_V) and were defined and calculated in accordance with the methods outlined in chapter 3.0 (sub-chapter 3.5).

8.3.4 *Indices of magnetically evoked neuromuscular performance*

The estimates of magnetically evoked neuromuscular performance of the knee flexors of the preferred leg were static peak twitch force (P_TF_E), electromechanical

delay (EMD_E) and rate of force development (RFD_E) and were defined and calculated in accordance with the methods outlined in chapter 3.0 (sub-chapter 3.6).

8.3.5 *Static fatiguing exercise task*

The static exercise task consisted of 30 seconds sustained maximal activation of the knee flexors of the preferred leg followed by a 5-second rest period and a further bout of 5 seconds effort.

8.3.6 *Eccentric exercise*

Following habituation and a warm-up of 5 sub-maximal and 5 maximal eccentric activations of the knee flexors of the preferred leg, participants performed 6 sets (each separated by 1 minute) of 10 repetitions of maximal eccentric activations on an isokinetic dynamometer (Kin-Com, Chattecx, Chattanooga, USA) at an angular velocity of $60°.s^{-1}$ ($1.05\ rad.s^{-1}$) through a range of 70° to 10° knee flexion (0° = full extension).

8.3.7 *Indirect markers of muscle damage*

Prior to the evaluation of performance on each assessment occasion, subjective assessments of soreness of the knee flexors of the preferred leg and blood samples were obtained. Participants were required to rate their soreness on a 100 cm visual analogue scale (Gleeson et al., 2003) on stretching and active flexion of the knee of the preferred leg. The statements on the scale read: 'my muscles don't feel sore at all', 'my muscles feel sore when I move them' and 'my muscles feel so sore that I don't want to move them', which corresponded to numerical ratings of 0, 5 and 10, respectively. Blood samples were obtained by means of capillary punctures to

assess the concentration of creatine kinase (CK), routinely used as an indirect indicator of muscle ultrastructural integrity (Vincent and Vincent, 1997). The finger was cleaned using an alcohol swab. The initial sample of blood was removed and a 32μl sample collected into a capillary tube and immediately pipetted onto a test strip for analysis. Creatine kinase concentration was analysed using a calorometric assey procedure (Reflotron, Boehringer Manheim, UK) and recorded in U/L. In the event of CK concentrations exceeding the recordable range (> 2300 U/L), 32μl dilutions of purified water were applied to the sample and the subsequent value was corrected accordingly. Based on the large inter-subject variability of CK measurements following muscle damage and recovery (Vincent and Vincent, 1997), CK values were subjected to logarithmic transformations for statistical analysis (CK_{log}). This approach has been used previously (Brown et al., 1997).

8.3.8 *Statistical analyses*

The selected performance indicators were described using ordinary statistical procedures (mean ± SD). The effect of the eccentric exercise intervention on performance subsequent to the static fatiguing exercise task over time was evaluated for each index of performance using separate three (condition: CON1; CON2; EIMD) by six (time: pre; 1h; 24h; 48h; 72h; 168h) by two (static fatiguing exercise: pre; post) fully repeated measures ANOVAs. Comparisons between muscle groups (knee flexors, knee extensors) of the dominant leg following eccentric exercise were evaluated for each index of performance by separate two (condition: CON; EIMD) by six (time: pre; 1h; 24h; 48h; 72h; 168h) by two (muscle group: knee flexors;

knee extensors) fully repeated measures ANOVAs, where CON represents the mean of scores for CON1 and CON2 obtained prior to the static fatiguing exercise task.

Creatine kinase levels and perceived soreness were assessed by means of separate three (condition: CON1; CON2; EIMD) by six (time: pre; 1h; 24h; 48h; 72h; 168h) fully repeated measures ANOVAs.

The assumptions underpinning the use of repeated measures ANOVA were checked and violations corrected by the Greenhouse-Geisser adjustment of the critical F-value, as indicated by $_{GG}$. Statistical significance was accepted at $p < 0.05$.

The experimental design offered an approximate .80 power of avoiding a Type-II error when employing a least detectable difference of 16 N, 8 ms, 350 N.s-1 and 12 ms for PF_V, EMD_V, RFD_V and $T_{½V}$ respectively and 3.13 N, 3.5 ms, 208 N.s-1 and 1.1 ms for P_TF_E, EMD_E, RFD_E and $T_{½E}$ respectively.

8.4 Results

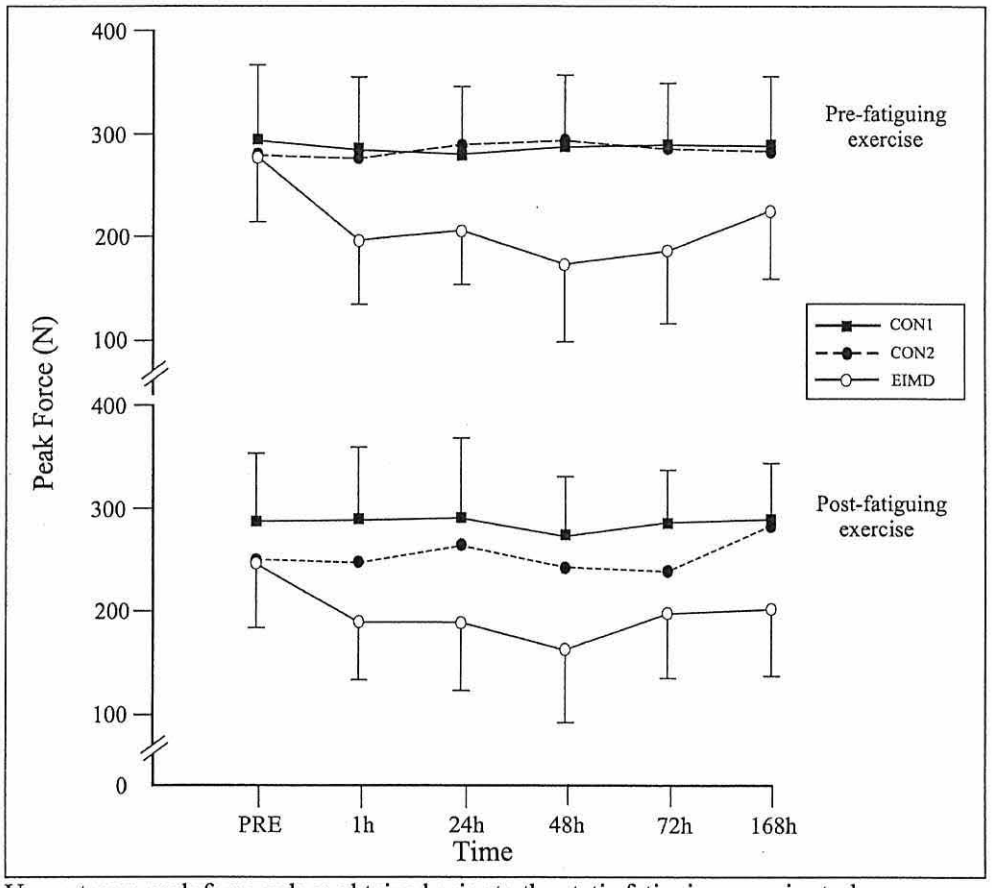
8.4.1 Preferred leg: knee flexors

8.4.1.1 *Volitional muscle activation*

Indices of volitional peak force (PF_V)

A significant two-factor condition (CON1; CON2; EIMD) by time (pre; 1h; 24h; 48h; 72h; 168h) interaction associated with the repeated measures ANOVA showed that while absolute PF_V performance was preserved during both control tasks (CON1, CON2), the EIMD condition was associated with a reduction in absolute PF_V (group mean ± SD) performance compared to baseline scores ($F_{[10, 60]} = 4.6$, $p < 0.001$) that was most prominent at 48h following eccentric exercise (276.2 ± 61.5 N vs. 197.5 ± 58.1 N, 204.6 ± 76.6 N, 172.6 ± 83.0 N, 184.3 ± 75.7 N and 226.5 ± 66.6 N, respectively). The actual losses to PF_V performance were 28.5%, 25.9%, 37.5%, 33.3% and 18.0% at 1h, 24h, 48h, 72h, 168h, respectively, compared to pre-eccentric exercise levels (please see table 8.1 and figure 8.2). A significant two-factor condition (CON1; CON2; EIMD) by fatiguing static exercise (pre; post) interaction associated with the repeated measures ANOVA showed that while absolute PF_V performance was preserved during the control task CON1, the static exercise task elicited fatigue, characterised by a similar level of reduction in PF_V performance in both CON2 (mean 10.1%) and EIMD (mean 6.3%) conditions ($F_{[2, 12]} = 8.6$, $p < 0.001$). These results suggest that the eccentric exercise induced a significant and prolonged loss in PF_V capabilities of the knee flexors of the dominant limb, however, the EIMD intervention did not substantively influence the subsequent effects of the fatiguing static exercise task (please see figure 8.2).

Figure 8.2. Peak force (PF_V) performance subsequent to volitional activation of the knee flexors over the three treatment conditions (group mean ± SD) (some SD bars removed for clarity).

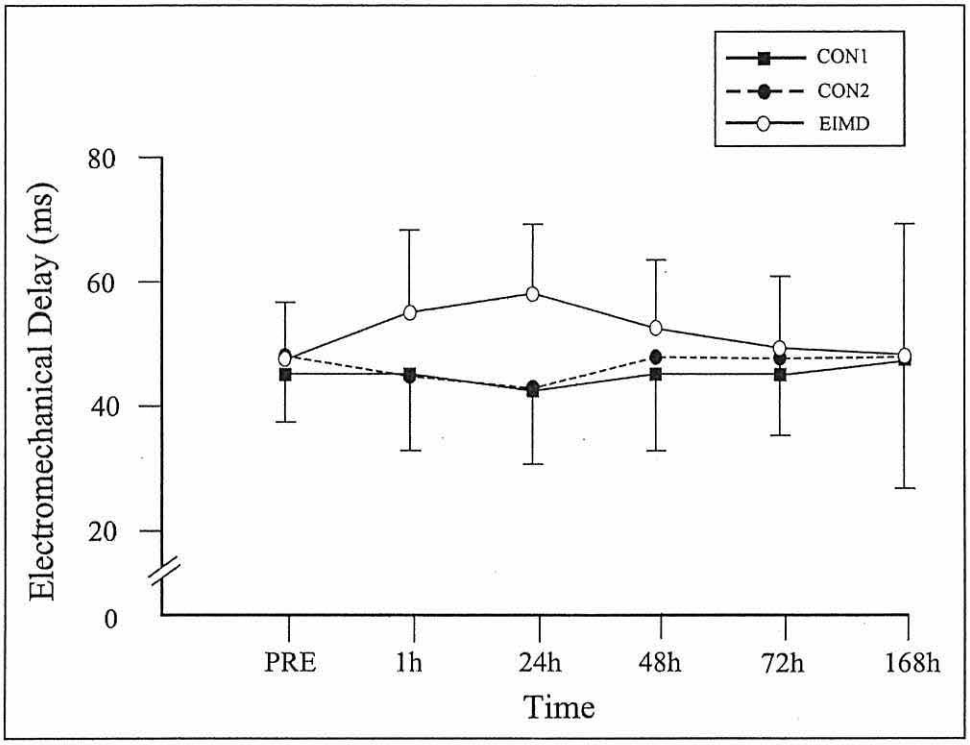


Upper trace: peak force values obtained prior to the static fatiguing exercise task
Lower trace: peak force values obtained immediately following the static fatiguing exercise task.

Indices of volitional electromechanical delay (EMD_V)

Despite a trend towards prolonged EMD_V following EIMD compared to baseline values (up to 23.9% at 24h), none of the interactions associated with the repeated measures ANOVA reached significance. A significant main effect for static fatiguing exercise ($F_{[1, 6]} = 8.2$, $p < 0.05$) indicated that averaged EMD_V performance (pre- and post- fatiguing exercise) associated with this condition was generally prolonged compared to the other treatment conditions, however the analyses were unable to distinguish differences over time (please see figure 8.3).

Figure 8.3. Electromechanical delay (EMD_V) performance subsequent to volitional activation of the knee flexors over the three treatment conditions (group mean ± SD) (some SD bars removed for clarity).

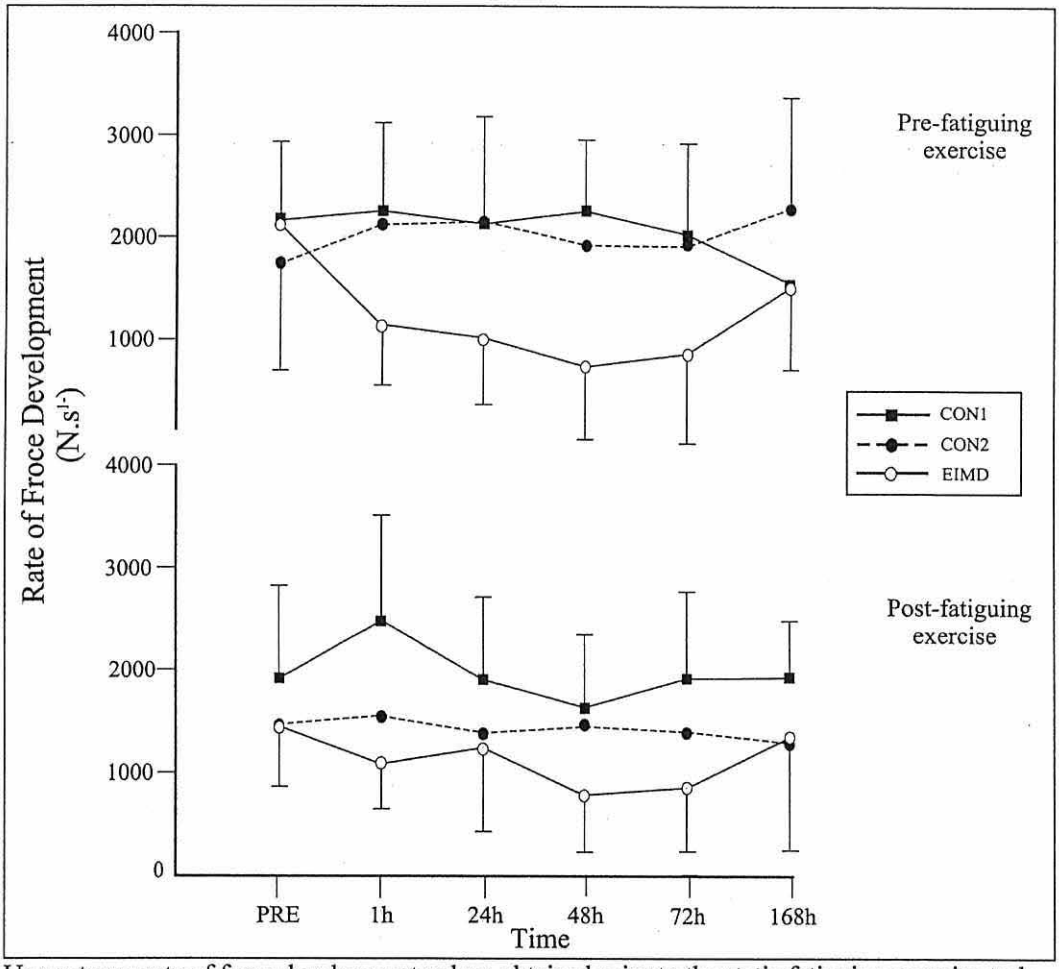


Indices of volitional rate of force development (RFD_V)

A significant two-factor condition by time interaction associated with the repeated measures ANOVA showed that while absolute RFD_V performance was preserved during both control tasks (CON1, CON2), the EIMD condition was associated with a reduction in RFD_V performance compared to baseline scores ($F_{[10, 60]} = 2.8$, $p < 0.01$) that was most prominent at 48h following eccentric exercise. The actual losses to RFD_V performance were 39.3%, 51.3%, 65.2%, 59.6% and 30.5% at 1h, 24h, 48h, 72h, 168h, respectively, compared to pre-eccentric exercise levels (please see figure 8.4). A significant two-factor condition by static fatiguing exercise (pre, post) interaction associated with the repeated measures ANOVA showed that while absolute RFD_V performance was preserved during the control task CON1, the performance of the static exercise task elicited average performance reductions of

36.7% and 5.8% (group mean values) in RFD_V performance compared to pre-fatigue levels in CON2 and EIMD conditions, respectively ($F_{[2, 12]} = 4.3$, $p < 0.05$) (please see figure 8.4)..

Figure 8.4. Rate of force development (RFD_V) performance subsequent to volitional activation of the knee flexors over the three treatment conditions (group mean ± SD) (some SD bars removed for clarity).



Upper trace: rate of force development values obtained prior to the static fatiguing exercise task
Lower trace: rate of force development values obtained immediately following the static fatiguing exercise task.

Table 8.1. Group mean scores for indices of volitional neuromuscular performance of the knee flexors associated with the exercise induced muscle damage (EIMD) treatment condition prior to (a) and immediately following (b) the static fatiguing exercise task (mean ± SD).

Index			Time					
			Pre	1h	24h	48h	72h	168h
PF_V	(N)	a	276.2 ± 61.5	197.5 ± 54.8	204.6 ± 71.6	172.6 ± 83.0	184.3 ± 75.7	226.5 ± 66.6
		b	247.7 ± 60.0	182.0 ± 55.0	183.5 ± 64.7	168.5 ± 70.0	192.6 ± 66.0	200.4 ± 67.4
EMD_V	(ms)	a	47.3 ± 9.7	54.6 ± 14.0	58.7 ± 13.9	52.0 ± 11.2	48.1 ± 13.3	50.7 ± 22.4
		b	47.6 ± 13.5	60.5 ± 26.5	67.3 ± 32.0	68.6 ± 28.4	62.6 ± 31.5	69.7 ± 39.8
RFD_V	($N.s^{-1}$)	a	2178 ± 878	1321 ± 516	1059 ± 620	758 ± 784	879 ± 942	1514 ± 876
		b	1436 ± 528	1071 ± 447	1235 ± 810	761 ± 513	782 ± 662	1409 ± 1117

8.4.1.2 *Magnetically evoked muscle activation*

Indices of magnetically evoked peak twitch force (P_TF_E)

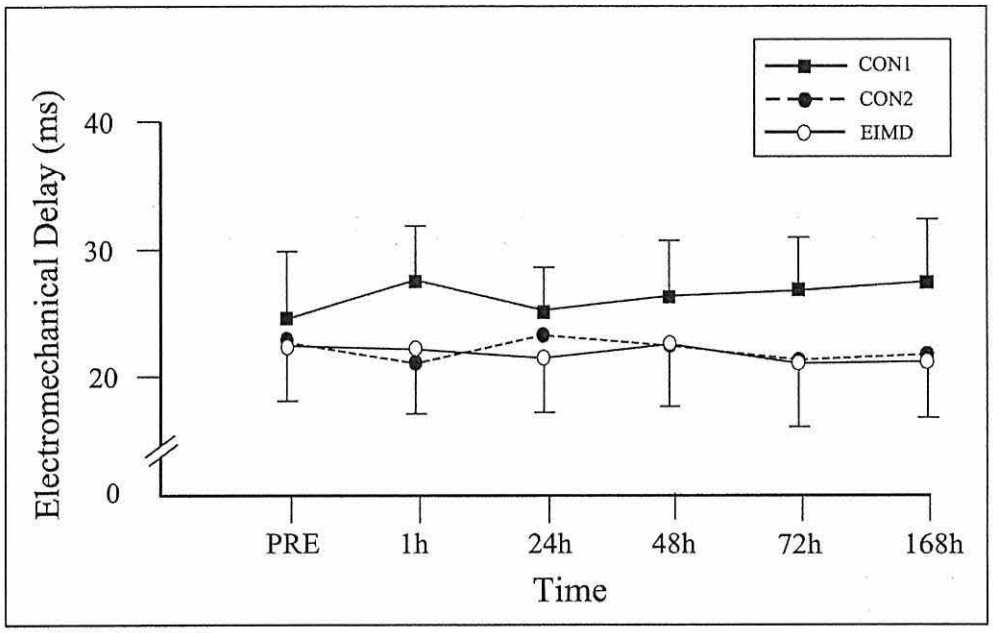
There were no significant interaction effects associated with the repeated measures ANOVA. A significant main effect for static fatiguing exercise ($F_{[1, 6]} = 28.8$, $p < 0.01$) indicated that P_TF_E performance associated with post measures was superior than compared to pre measures.

Indices of magnetically evoked electromechanical delay (EMD_E)

A significant condition by static fatiguing exercise interaction revealed that while absolute EMD_E performance was preserved during the control task CON1, performance of the static fatiguing exercise task elicited an average group mean improvement in EMD_E performance in CON2 and EIMD conditions of 13.0% and 15.7%, respectively, compared to pre-fatigue values (25.8 ± 1.0 ms vs. 22.4 ± 1.0 and 25.8 ± 0.60 ms vs. 21.7 ± 0.60 ms, respectively, $F_{[2, 12]} = 8.6$, $p < 0.01$) (please see figure 8.5). These results suggest that eccentric exercise did not affect EMD_E

performance, or influence the physiologic response to the static fatiguing exercise task.

Figure 8.5. Effects of exercise-induced muscle damage on EMD_E performance of the knee flexors subsequent to a 35-second static fatiguing exercise task (group mean ± SD) (some SD bars removed for clarity).



Indices of magnetically evoked rate of force development (RFD_E)

Despite a trend towards improved RFD_E following static fatiguing exercise, none of the interactions associated with the repeated measures ANOVA reached significance. A significant main effect for static fatiguing exercise ($F_{[1, 6]} = 22.2$, $p < 0.01$) indicated that group mean averaged RFD_E performance associated with pre-'fatigue' measures was superior than compared to post-'fatigue' values, but analyses were not able to distinguish between treatment conditions.

Table 8.2. Group mean scores for indices of magnetically evoked neuromuscular performance of the knee flexors associated with the exercise induced muscle damage (EIMD) treatment condition prior to (a) and immediately following (b) the static fatiguing exercise task (mean ± SD).

Index			Time					
			Pre	1h	24h	48h	72h	168h
P_TF_E	(N)	a	34.1 ± 5.2	23.9 ± 5.8	27.9 ± 5.2	24.6 ± 6.6	28.8 ± 9.5	31.2 ± 5.0
		b	42.0 ± 16.0	34.8 ± 11.6	39.4 ± 9.6	33.3 ± 8.5	40.4 ± 14.0	39.5 ± 12.3
EMD_E	(ms)	a	26.2 ± 4.2	25.0 ± 4.3	25.6 ± 4.3	27.0 ± 5.0	25.0 ± 4.1	25.7 ± 3.9
		b	22.1 ± 3.3	22.2 ± 3.3	21.5 ± 2.6	22.4 ± 2.8	21.0 ± 2.4	21.1 ± 2.3
RFD_E	($N.s^{-1}$)	a	1139 ± 305	946 ± 253	1056 ± 261	924 ± 342	1158 ± 445	1230 ± 215
		b	1500 ± 648	1451 ± 552	1630 ± 614	1311± 518	1696 ± 712	1539 ± 601

The current results indicate that eccentric exercise did not induce impairments to magnetically evoked indices of neuromuscular performance. However, there was a significant and prolonged reduction in the volitional contractile capabilities of the knee flexors. Performance of the acute fatigue task induced further decrements to the volitional force generating capabilities of the knee flexors. Consistent with the previous studies (chapters 6 and 7), indices of magnetically evoked neuromuscular performance were not impaired following the static fatiguing exercise task, in fact EMD_E performance was potentiated.

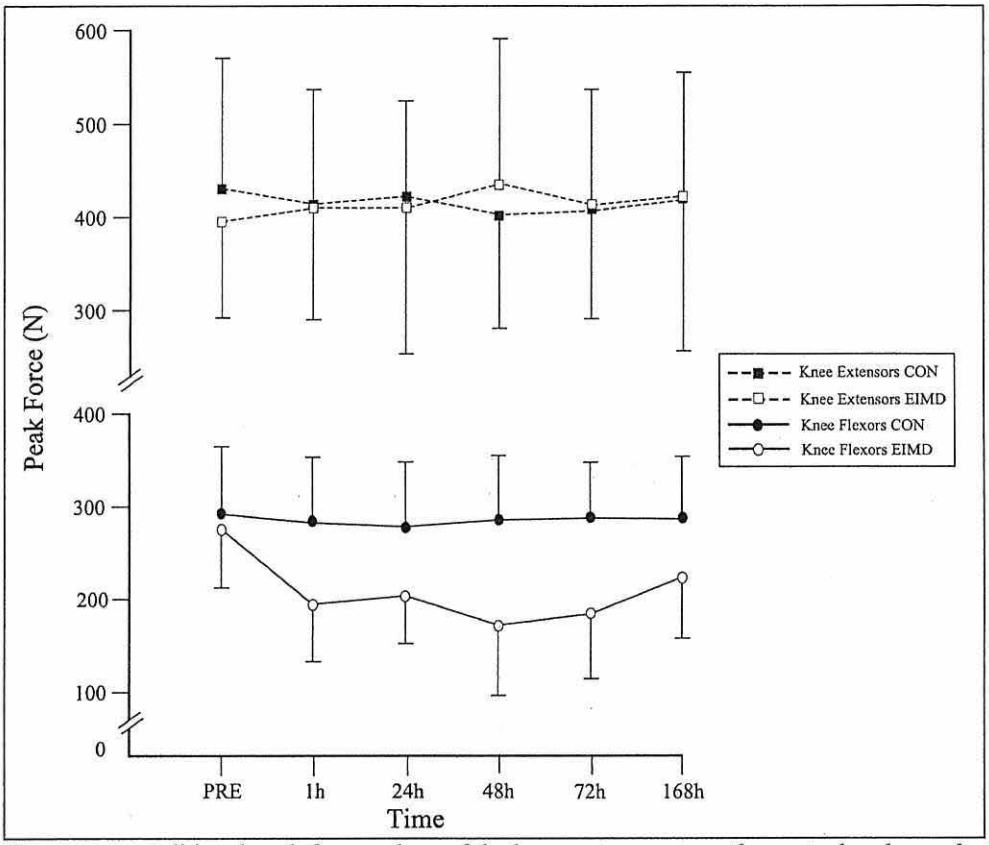
8.4.2 Preferred leg: knee flexor and extensor comparisons

8.4.2.1 *Volitional muscle activation*

Indices of volitional peak force (PF_V)

A significant three-factor condition (CON; EIMD) by time (pre; 1h; 24h; 48h; 72h; 168h) by muscle group (knee flexors; knee extensors) interaction associated with the repeated measures ANOVA ($F_{[5, 30]} = 5.2$, $p < 0.01$) suggested that PF_V performance was maintained during the control task for both muscle groups and the EIMD condition for the knee extensors. However, the EIMD intervention elicited a reduction in absolute PF_V performance, that peaked at 48h (37.5% decrease) compared to pre-eccentric exercise levels in the knee flexors (please see figure 8.6).

Figure 8.6. Peak force (PF_V) performance subsequent to volitional activation of the knee flexors and knee extensors over the two treatment conditions (group mean ± SD).



Upper trace: volitional peak force values of the knee extensors over the control and muscle damage conditions

Lower trace: volitional peak force values of the knee flexors over the control and muscle damage conditions.

Indices of volitional electromechanical delay (EMD_V)

A significant two-factor condition by muscle group interaction associated with the repeated measures ANOVA ($F_{[1, 6]} = 10.1$, $p < 0.05$) showed that while average (group mean ± SD) EMD_V performance levels associated with the control and EIMD conditions in the knee extensors were similar (33.0ms ± 1.2 ms vs. 32.0 ± 2.5 ms, respectively), the EIMD condition was associated with prolonged average (group mean ± SD) EMD_V scores in the knee flexors (45.9 ± 2.1 ms vs. 51.9 ± 4.2 ms, respectively [13.0% increase]) compared to the control. The significant two-factor condition by time interaction associated with the repeated measures ANOVA

showed that while absolute EMD_V performance was preserved during the control, the EIMD condition was associated with an increase in absolute (group mean ± SD) EMD_V scores of 11.6%, 12.8% and 7.8% at 1h, 24h and 48h, respectively (40.0 ± 10.3 ms vs. 44.7 ± 13.9 ms, 45.1 ± 19.1 ms and 43.1 ± 12.6 ms, respectively, $F_{[5, 30]} = 2.7$, $p < 0.05$) compared to pre eccentric exercise values.

Indices of volitional rate of force development (RFD_V)

A significant three-factor interaction associated with the repeated measures ANOVA ($F_{[5, 30]} = 4.4$, $p < 0.01$) suggested that while RFD_V performance was maintained during the control task for both muscle groups, the EIMD intervention task elicited a reduction in RFD_V performance at all assessment points following the eccentric exercise (peaking at 48h [65.2% decrease]) compared to pre-eccentric exercise levels in the knee flexors, whereas absolute RFD_V performance in the knee extensors demonstrated impairments at 48h, 72h and 168h of a smaller magnitude (18.0%, 14.1% and 16.0% decreases, respectively).

Table 8.3. Group mean scores for indices of volitional neuromuscular performance of the knee extensors associated with the exercise induced muscle damage (EIMD) treatment condition (mean ± SD).

Index		Time					
		Pre	1h	24h	48h	72h	168h
PF_V	(N)	395.6 ± 107.5	410.1 ± 124.7	411.9 ± 113.5	431.1 ± 167.7	414.9 ± 120.0	423.8 ± 138.4
EMD_V	(ms)	32.7 ± 7.6	34.9 ± 10.0	31.6 ± 9.8	34.2 ± 13.1	28.5 ± 6.0	30.0 ± 9.2
RFD_V	($N.s^{-1}$)	2841 ± 1606	2774 ± 1593	2622 ± 1289	2384 ± 1411	2740 ± 1730	2388 ± 1775

8.4.2.2 *Magnetically evoked muscle activation*

Indices of magnetically evoked peak twitch force (P_TF_E)

None of the interactions associated with the repeated measures ANOVA reached significance. A significant main effect for muscle group ($F_{[1, 6]} = 27.7$, $p < 0.01$) indicated that group mean P_TF_E performance scores associated with the knee extensors were superior compared to the knee flexors in both the control and EIMD conditions. These results suggest that the EIMD condition did not influence the P_TF_E performance of either muscle group.

Indices of magnetically evoked electromechanical delay (EMD_E)

None of the interactions associated with the repeated measures ANOVA reached significance. A significant main effect for muscle group ($F_{[1, 6]} = 106.9$, $p < 0.001$) indicated that group mean EMD_E performance scores associated with the knee extensors were superior compared to the knee flexors in both the control and EIMD conditions. These results also suggest that the EIMD condition did not influence the EMD_E performance capacity of either muscle group.

Indices of magnetically evoked rate of force development (RFD_E)

Repeated measures ANOVA revealed no significant interactions or main effects.

Table 8.4. Group mean scores for indices of magnetically evoked neuromuscular performance of the knee extensors associated with the exercise induced muscle damage (EIMD) treatment condition (mean ± SD).

Index		Time					
		Pre	1h	24h	48h	72h	168h
P_TF_E	(N)	74.8 ± 23.7	68.1 ± 24.6	66.0 ± 20.5	68.6 ± 19.3	77.9 ± 24.2	75.4 ± 26.7
EMD_E	(ms)	13.5 ± 1.2	13.4 ± 1.6	13.8 ± 1.5	13.2 ± 1.1	13.1 ± 1.6	13.9 ± 1.3
RFD_E	($N.s^{-1}$)	1303 ± 286	1256 ± 361	1236 ± 395	1287 ± 466	1483 ± 452	1326 ± 371

These results show that in general, the knee extensors are associated with significantly superior absolute neuromuscular performance subsequent to volitional and magnetically evoked muscle activation, compared to the knee flexors. The effects of the EIMD intervention on the performance capabilities of knee extensors were minimal and were perhaps confounded by the heterogeneity of group responses.

8.4.3 Indirect indicators of muscle damage

8.4.3.1 *Creatine kinase*

Repeated measures ANOVA of CK_{log} values revealed a significant two-factor condition by time interaction, which showed that while CK_{log} levels remained constant over both control conditions (CON1; CON2), the EIMD condition was associated with an increase in group mean values commencing at 24h, which was most prominent at 72h following eccentric exercise ($F_{[10, 60]} = 15.3$, $p < 0.001$) (please see table 8.5).

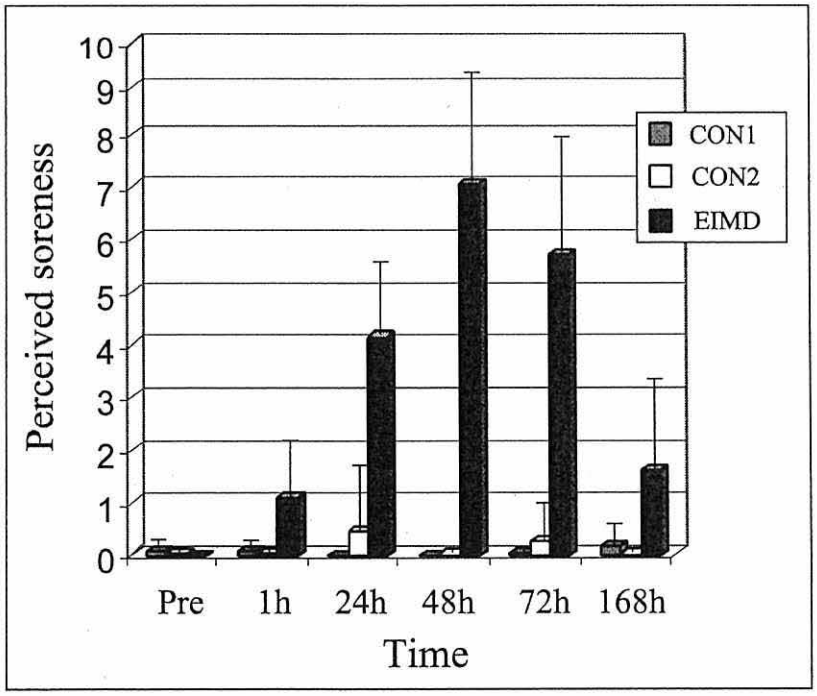
Table 8.5. Group mean scores of back transformed CK_{log} values associated with the exercise induced muscle damage (EIMD) treatment condition (± 95% confidence limits).

Index		Time					
		Pre	1h	24h	48h	72h	168h
CK	(U/L)	93.3 ± 2.76	117.5 ± 3.0	275.4 ± 3.55	1117.9 ± 8.55	2754.2 ± 13.35	1259.0 ± 7.55

8.4.3.2 *Perceived soreness*

A significant two-factor condition by time interaction associated with the repeated measures ANOVA showed that while perceived soreness remained constant over both control conditions (CON1; CON2), the EIMD condition was associated with an increase in group mean values commencing at 24h, which was most prominent at 48h following eccentric exercise ($F_{[10, 60]} = 7.0$, $p < 0.001$) (please see figure 8.7).

Figure 8.7. Ratings of perceived soreness of the knee flexors over the three treatment conditions (group mean ± SD).



8.5 Discussion

The absence of change over the CON1 treatment condition for each index of performance indicates that there were no systematic or learning effects and that observed changes in performance throughout the other treatment conditions can be attributed to the associated exercise intervention(s). The potential decrements to neuromuscular performance caused by muscle fatigue have previously been interpreted to represent an increased risk of injury (Gleeson et al., 1998; Mercer et al., 1998; Chan et al., 2001). The potential for prolonged impairment to neuromuscular performance capabilities following eccentric exercise-induced muscle damage (Byrne et al., 2001; Nokasa et al., 2001) may, therefore, be associated with a prolonged increased risk of injury. The purpose of the EIMD condition in the present study was to investigate the effects of muscle damage on volitional and magnetically evoked neuromuscular performance and the subsequent dynamic capabilities for knee joint protection. The inclusion of a fatigue trial enabled the investigation of any interaction effects of EIMD and acute muscle fatigue and the establishment of the potential relative impact of each of the different exercise stresses on protective neuromuscular performance capabilities.

8.5.1 Knee flexors:

8.5.1.1 *Effects of eccentric exercise*

The current results strongly suggest that the eccentric exercise protocol induced muscle damage, characterised by an increase in serum CK values, most prominent at 72h and ratings of perceived soreness, consistent with the DOMS phenomenon, commencing at 24h and peaking at 48h. In addition, whereas performance over the control conditions showed no change, a significant and prolonged decrease in PF_V of the knee flexors of the preferred leg was observed (please see figure 8.2), of a similar level to the knee extensors previously observed in this laboratory (Byrne et al., 2001; Marginson, 2003 unpublished Ph.D. thesis) following similar exercise protocols. These results are also consistent with other investigations of the lower extremities (McHugh et al., 2000; Brockett et al., 2001). The current decrement to PF_V of the knee flexors following eccentric exercise is likely to reflect a complex interaction of several factors. These may include possible contributions of structural damage to a proportion of muscle fibres and associated organelles (Hortobagyi et al., 1998; Stupka et al., 2001), possible disruption to intra-muscular Ca^{2+} dynamics (Morgan and Allen, 1999), increased recruitment of slow-twitch muscle fibres (Warren et al., 2000) and potential conscious/unconscious inhibition as a result of sensitisation of nocioceptors (Latash, 1998) associated with the pain response. However, despite this, no significant changes were observed to EMD_V performance capabilities. The aforementioned processes in addition to possible deterioration of muscle fibre conduction velocity (Linnamo et al., 2000) might be expected to induce impairment to the temporal capability of the musculature, particularly considering the extent of PF_V performance loss. However, the moderate power associated with this analysis (0.63) may suggest the possibility of a

Type II error and limited capability to discriminate subtle changes in performance. It is also plausible that the lack of significant change to EMD_V performance may reflect the effects of other changes within the muscle opposing the reduced contractile efficiency. For example, some researchers report a shift in the length-tension relationship towards longer muscle lengths (Morgan and Allen, 1999; Brockett et al., 2001). This adaptation is believed to be due to a shortening of some sarcomeres as compensation for the over stretched and irreversibly damaged sarcomeres in series following eccentric exercise (Morgan and Allen 1999). Indirect observations of this phenomenon are derived from a greater relative loss to peak force capabilities at short compared to long muscle lengths. This was observed in a pilot investigation prior to this experiment, whereby a greater relative loss to peak force capabilities at 80° compared to 25° knee flexion was noted (248.6 ± 60.6 vs. 170.3 ± 72.5 [31.5% decrease] and 208.8 ± 34.8 vs. 99.9 ± 25.7 [52.2% decrease], respectively 48 hours following exercise) (Williams, 2002, unpublished Masters Thesis). Since the elastic component in series with the contractile component can be stretched by a shortening of the involved muscle, the shortening of some muscle fibres to compensate for the damaged sarcomeres caused by EIMD, may elicit a greater residual stretch of the SEC. In addition, possible intra-muscular swelling reported by several investigations following eccentric exercise (Foley et al., 1999; Brockett et al., 2001), may also induce lengthening of the SEC by distension of the involved musculature. Given that the majority of the EMD is determined by the time taken to elongate the SEC (Norman and Komi, 1979), such changes subsequent to exercise-induced muscle damage may be sufficient to counter any EMD_V performance decrements associated with reduced contractile

performance. However, further investigation would elucidate more fully the potential of effects of these processes on EMD.

Results for volitional rate of force development (RFD_V) performance revealed substantive decrements to the temporal capability to generate muscle force following eccentric exercise, of up to 65.2% at 48h. Coupled with the impairment to PF_V performance, this may present substantive challenges to the neuromuscular system of the knee flexors to generate meaningful levels of force in response to dynamic loading of the knee and accordingly, preserve joint integrity. This may be particularly true considering that such mechanical disruption of muscle fibres resulting from exercise-induced muscle damage may also severely jeopardise proprioceptive capabilities (Saxton et al., 1995). However, maintenance of the capability to initiate muscle force (EMD_V) may be a part of vital neuromuscular compensatory mechanisms enabling protection of the joint system in emergency scenarios. For example, the shift of optimal force production towards longer muscle lengths (Morgan and Allen, 1999; Brockett et al., 2001) in addition to potential facilitatory effects to EMD_V by stretching the SEC, may also aid in the conservation of neuromuscular performance at joint angles where key ligamentous structures are under greatest mechanical strain. Furthermore, the observed pain response, which peaked in conjunction with maximal decrements volitional neuromuscular performance, may cue reduced activity levels during conditions of compromised dynamic neuromuscular capabilities. Certain compensatory strategies to limit further damage and the potential for joint injury may be deployed following eccentric exercise-related impairment to the contractile capabilities of the knee flexors. These proceses may enable a level of joint protection at unfavourable knee

angles, or indeed, provoke the avoidance of situations that may involve intense dynamic joint loading. Reference to magnetically evoked responses may reveal whether or not these impairments to volitional neuromuscular performance are associated with an overall decrease in neuromuscular performance capacity.

Despite the proposed increased susceptibility of fast-twitch fibres to damage following eccentric exercise (Friden and Lieber, 1992; Linnamo et al., 2000; Brockett et al., 2002), the current results show that the physiologic capacity of the neuromuscular system, as measured by twitch responses to magnetic stimulation, was not impaired subsequent to the eccentric exercise protocol. Although it is suggested that during maximal eccentric activation of muscle, fast-twitch motor units may be preferentially recruited (McHugh et al., 2000; 2002), the current results may suggest that under volitional, non-emergency conditions, protective inhibitory processes may restrict access to the full capacity of fast twitch high threshold motor units. Accordingly a 'reserve' capacity of high threshold units may be retained, potentially available to the sports performer when a true emergency scenario is perceived. It is acknowledged that other factors may account for this observation, the most simple being that participants refrained from exerting maximal effort during periods of volitional muscle activation by means of potential conscious or subconscious pacing strategies. In an attempt to minimise this, verbal encouragement was given during periods of MVMA, commensurate with the prior investigations in this thesis. Similarly, Strojnik and Komi (2000) reported no impairment to the electrically evoked EMD of the knee extensors subsequent to a prolonged stretch-shortening inclined sledge jumping protocol, however, unlike the present study, they reported a reduction in peak twitch force. The decrease in peak

twitch force observed by Strojnik and Komi (2000) may have been due to a combination of the effects of an 'amount' of muscle damage in addition to contractile failure caused by metabolic changes due to the prolonged nature of the task. Furthermore, while eccentric muscle activation is associated with an 'unloading' of the SEC (Jones et al., 1989), the concentric component of the aforementioned stretch-shortening protocol may have been associated with stress relaxation of the SEC (Stone, 1992), which may have countered any potential EMD changes due to contractile failure. These factors, combined with other potential adaptations following exercise-induced muscle damage may account for the 'preservation' of EMD performance at a fixed muscle length, yet explain the observation of decreased contractile performance. Some of these adaptations, such as EIMD-induced muscle length changes (Morgan and Allen, 1999; Brockett et al., 2001), may well be apparent in the current investigation. However, the most important observation from the current study is that even when the stress of exercise is elevated to a level that might be associated with mechanical damage to the involved musculature, the neuromuscular system is still able to preserve a capacity to respond that is analogous to muscle asymptomatic of injury. This is evident by the parity in neuromuscular performance capacities as measured by magnetic stimulation during EIMD and control conditions. These findings may, therefore, offer further evidence of a 'reserve' capacity of large, high threshold motor units capable of protecting the joint system against injury. In the presence of substantive decrements to volitional performance capabilities, such neuromuscular compensatory processes may, indeed, be integral to the avoidance of knee injury. However, the utility of this mechanism is entirely dependent on whether the protective inhibitory processes that restrict volitional access to the total capacity of

the musculature (Hopkins and Ingersoll, 2000) can be overcome, or that the extent of the 'emergency' is recognised properly by the individual so that appropriate and timely responses can be marshalled.

8.5.1.2 *Effects of an acute static fatiguing exercise task*

Some research exists documenting the premature onset of fatigue in muscle symptomatic of damage compared to healthy muscle, characterised by a greater accumulation of blood lactate during incremental dynamic exercise (Gleeson et al., 1995). However, little is known about the neuromuscular responses to fatiguing exercise following muscle damage. The current results show that the static exercise task induced fatigue in the knee flexors, characterised by an immediate loss to PF_V performance, of a similar magnitude across both treatment conditions, which did not vary systematically across the time period of EIMD monitoring (pre to 168h) (average group mean percentage changes of 10.1% and 6.3% during CON2 and EIMD, respectively). The current level of fatigue is similar to the impairment to PF_V performance observed in males in chapter 6.0 (15.0%) following a similar exercise protocol and is also congruent with the extent of performance loss as a result of match play in team games such as soccer (Gleeson et al., 1998b). Additionally, decreases in RFD_V performance were also observed following fatigue, which were greater in the CON2 compared to the EIMD condition, however, such did not vary systematically over the time period of EIMD monitoring (pre to 168h) (average group mean percentage decreases 36.7% and 5.8% for CON2 and EIMD, respectively). The current results show that despite the reduced absolute performance levels following prior eccentric exercise, the performance of the static

fatiguing exercise task was associated with further reductions to the contractile capabilities of musculature symptomatic of damage. For example, the net effect of PF_V impairments of up to 37.5% (at 48h) coupled with an approximate 6.3% further reduction following acute maximal exercise may render peak force performance capabilities of just under half (40%) that of healthy muscle prior to exercise (please see figure 8.2). This is also evident in the case of RFD_V at 1h, 72h and 168h. Such impairment to the temporal capability to generate meaningful levels of force, even in the absence of significant changes to EMD_V performance, may present substantive challenges to the capability of the knee flexors to maintain knee joint integrity in response to dynamic loading. However, given that the peak impairments to the force generating capabilities (at 48 hours) following EIMD were far in excess of the average impairments to these indices of performance subsequent to an acute fatigue task (decreases: PF_V 37.5% vs. 10.1%, RFD_V 65.3% vs. 36.7% following EIMD and fatigue, respectively), exercise leading to muscle damage may have greater relative impact on knee injury avoidance capabilities compared to acute muscle fatigue. Interestingly, despite such impairment to the volitional performance capabilities of the knee flexors, magnetically evoked indices of performance were not impaired following fatigue in either healthy, or muscle symptomatic of damage. In fact, consistent with the previous studies (chapter 6, chapter 7), EMD_E was improved, by similar amounts in both treatment conditions (13.0% vs. 15.7% during CON2 and EIMD, respectively [group mean average percentage changes]). These results suggest a remarkable preservation of neuromuscular capacity under conditions of acute muscle fatigue even in the presence of suspected mechanical disruption to the musculature. The most pertinent question to the sports performer may, therefore, be if the symptoms of pain (which

may peak commensurately with maximal decrements to performance (Brown et al., 1997; Rinard et al., 2000)) are ignored, would participation in sports activities result in a real increased likelihood of injury? Clearly, further investigation is required, however, the current results do present reasonable evidence to suggest that exercise-induced muscle damage and associated impairment to some indices of volitional neuromuscular performance, does not preclude the individual's ultimate ability to recruit certain compensatory mechanisms, which may counter an associated possible increased risk of injury.

8.5.2 Knee Extensors

The results indicate generally superior absolute neuromuscular performance levels associated with the knee extensors compared to the knee flexors. This finding is consistent with previous research (e.g. Gleeson and Mercer, 1992). However, the effects of EIMD on the agonist (knee flexors) on the volitional neuromuscular capabilities of antagonist muscle group (knee extensors) was limited to a minor decrease in RFD_V capabilities of up to 18.0% (at 48h) compared to pre-EIMD levels. While the mechanisms mediating this change in performance of the antagonist are currently unclear, perhaps the sensation of pain in the agonist, which was also most prominent at 48h, may have consciously or sub-consciously affected the force generating capabilities of the antagonist. Potential central adaptations to EIMD, however, may be expected to facilitate antagonist neuromuscular performance, for example, Leger and Milner (2001) reported that following eccentric exercise of the wrist extensors, an increase in antagonist coactivation was observed during sustained sub-maximal activation of the agonists. While it is

conceivable that eccentric exercise-induced disruption to the muscle spindles of the agonist and stimulation of nocioceptors (Latash, 1998), associated with the pain response following muscle damage (Brockett et al., 2001) may elicit decreased reciprocal inhibition (McComas, 1996; Leger and Milner, 2001), no significant improvements to antagonist (knee extensor) volitional neuromuscular performance were observed in the current study. Further investigation using a larger sample may offer greater experimental design sensitivity and a capability to elucidate potentially subtle changes in antagonist performance. Perhaps an important consideration given the limited impairment to neuromuscular performance of knee extensors during concomitantly impaired knee flexor performance capabilities is the potential for greater strain in the ACL. Unregulated activation of the knee extensors at vulnerable joint angles may be associated with greater anterior tibio-femoral shear forces and an increased susceptibility to ACL injury (Aune et al., 1995).

Magnetically evoked indices of performance indicated no change to the ultimate neuromuscular capacity of the knee extensors following eccentric exercise of the knee flexors.

8.5.3 *Implications for the sports performer*

In summary, the eccentric exercise protocol was associated with impaired PF_V and RFD_V performance of the knee flexors and a preserved temporal capability to initiate muscle force (EMD_V). Such changes to the maximal force generating capabilities of the knee flexors may present substantive challenges to the neuromuscular system to protect the ACL during mechanical loading of the knee

joint, especially at knee angles proximal to full extension. However, a potential shift in the length-tension relationship towards longer muscle lengths (Morgan and Allen, 1999), which presently corresponds to knee angle proximal to full extension, may be associated with preservation of neuromuscular performance at vulnerable joint positions. In addition, the maintenance of neuromuscular performance capacity, as measured by magnetic stimulation, following suspected muscle damage, may suggest a 'pool' of motor units remain as a 'reserve', un-recruited routinely during routine 'non-emergency' scenarios. If these magnetically evoked responses are representative of the neuromuscular performance capabilities available to an individual during 'emergency' threats to knee joint integrity, then such may afford a level of protection sufficient to avoid injury. Remarkably, during EIMD this capacity was maintained and even potentiated, in the case of EMD_E, following fatigue, where some indices of volitional performance experienced further impairment. This preservation of performance capacity may be a vital neuromuscular strategy to help prevent the sports performer becoming injured on each occasion when they experience fatigue and/or symptoms of exercise-induced muscle damage. Further study is required to investigate the potential effects of EIMD on the performance capabilities of the reciprocal muscle group and the likely implications for dynamic knee joint stability.

8.5.4 *Conclusions*

At knee angles where key ligamentous structures are under greatest mechanical strain, substantive impairments to the volitional capability of the knee flexors may place the sports performer at increased risk of injury. The current results, however,

suggest the deployment of neuromuscular compensatory strategies in response to impairments to volitional neuromuscular performance capabilities following eccentric exercise. These may include the pain response (DOMS), muscle length modifications (Morgan and Allen, 1999), possible preservation of EMD_V performance and the maintenance and potentiation (EMD_E) of the neuromuscular performance capacity following fatigue. These results suggest the potential for improved dynamic protection if possible inhibitory processes, which restrict access to the full capacity of motor units, can be 'down-regulated' during critical periods of joint loading.

Chapter 9
Final Discussion and Conclusions

9.0 FINAL DISCUSSION AND CONCLUSIONS

The aims of this thesis were: to establish the kinanthropometric utility associated with the selected indices of neuromuscular performance to be employed throughout these series of investigations; to evaluate effects of acute exercise stresses (including fatiguing exercise and EIMD) on the volitional neuromuscular performance capability and magnetically evoked performance capacity of the knee flexors and how these performance characteristics may vary between sexes and; to examine neuromuscular recovery following maximal intensity exercise and the possible effects of EIMD on the performance capability of the antagonist muscle group. These aims were chosen to further the understanding of performance changes associated with stressful exercise and concomitant injury-related threats to the integrity of synovial joints. These aims were investigated by means of five empirical investigations. The following sections will outline how such aims were examined and detail any new contributions to the literature.

9.1 New contributions to the literature

9.1.1 *Measurement utility of indices of volitional and magnetically evoked neuromuscular performance*

The data presented in studies 1 and 2 (chapters 4 and 5) showed parity in the measurement reproducibility characteristics of indices of volitional and magnetically evoked performance of the knee flexors, both during intra-session and inter-day assessments and under conditions of fatigue and un-fatigued muscle. This indicated that similar judgements could be made concerning protocol design for the assessment of neuromuscular performance. In addition, comparisons of the

modes of muscle activation showed that magnetically evoked indices of neuromuscular performance generally offered statistically equivocal levels of measurement reproducibility compared to traditional volitional methods. As such, the current data also lends support to this method of assessment based on its kinanthropometric utility. Furthermore, measurements acquired when men and women were either un-fatigued or fatigued offered similar levels of reproducibility and reliability and thus utility. The results of studies 1 and 2, however, indicated that apart from the index of volitional peak force, single trial protocols assessing all other indices of performance did not offer a level of measurement precision capable of detecting subtle changes (< 5%) in intra- or inter-individual performance. Based on the predicted number of replicates required to achieve this level of measurement sensitivity, the indices of volitional peak force (PF_V) and electromechanical delay (EMD_V) and magnetically evoked electromechanical delay (EMD_E) offered the greatest practical utility for the assessment of neuromuscular performance. Such indices required the mean of, for example, 1, 3 and 2 trials, respectively, during intra-session intra-group comparisons and 15, 26 and 25 trials, respectively, during inter-session intra-individual comparisons. The subsequent implications of these findings for contemporary clinical practice may mean that the accurate monitoring of the individual may be restricted to scenarios where fairly gross changes in performance capability are expected, such as during the preliminary stages of an ACL rehabilitation programme. The generally greater levels of measurement sensitivity afforded during intra-group compared to intra-individual contrasts may, however, facilitate the effective targeting of resources to individuals within a group of patients with greater requirements.

9.1.2 *Effects of exercise on neuromuscular performance; possible implications for anterior cruciate ligament (ACL) injury*

9.1.2.1 *Effects of acute muscle fatigue*

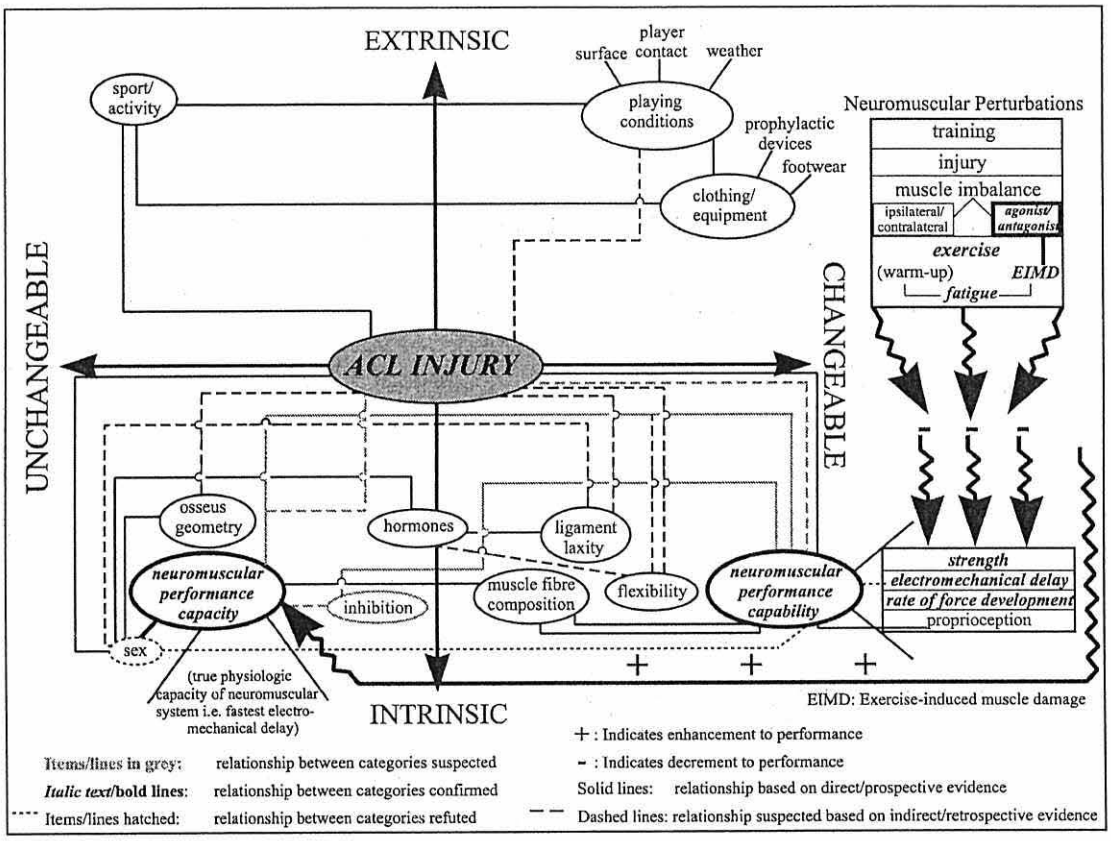
A conceptual model for knee joint stability dictates that optimal functioning of the knee flexors, in particular, is fundamental to the prevention of ACL injury (Johannson, 1991; Rees, 1994; Gleeson and Mercer, 1996). The neuromuscular reaction time (EMD) of this muscle group is integral to the protection of the joint system (Gleeson et al., 1998a; Mercer et al., 1998). However, this index of performance has received limited scrutiny in the knee flexors both under conditions of fatigued or un-fatigued muscle. Consideration of the processes that may compromise the temporal capabilities of the knee flexors may be particularly important with respect to the female athlete, given their increased susceptibility to ACL injury by comparison to male counterparts (Gray et al., 1985; Hutchinson and Ireland, 1995; Ireland, 1997). In addition, magnetic stimulation of the sciatic nerve, which may offer a truer estimate of knee flexor performance capacity (King and Chippa, 1989), may enable a greater understanding of the effects of fatigue on the capacity to avoid knee injury. The data obtained from study 3 (chapter 6) showed that acute maximal (static) exercise induced fatigue, identified by a decrease in PF_V performance. Study 4 (chapter 7) illustrated that such decrements to PF_V capabilities were cumulative subsequent to serial episodes of fatigue. Males experienced generally greater levels of fatigue by comparison to females in study 3, however, an increase in EMD_V latencies was only observed in females. While the mechanisms contributing to the increased fatigability of males is currently unclear, the observed changes in EMD_V performance may, in part, reflect differential compliance characteristics of the SEC between sexes (Winter and Brookes, 1991).

In the presence of concomitant decreases in force generating capabilities, this potential reduction in the dynamic protection of the joint may place the female athlete at greater risk of ACL injury, particularly considering other anatomical and biophysiological risk factors. Despite the near complete recovery of peak force by 6 minutes following cessation of exercise, EMD_V remained impaired throughout this time period. Given the potential importance of neuromuscular reaction time to knee joint stability (Gleeson et al., 1998b; Mercer et al., 1998), the lack of recovery of EMD_V capabilities of the knee flexors may be associated with a prolonged risk of injury should the performer recommence activity. Data from studies 3 and 4 show that magnetically evoked temporal responses (EMD_E, $T_{½E}$) in particular, demonstrated concomitant improvements throughout the period of decreased volitional capabilities. This implies the potential to initiate and muster muscle force much quicker than prior to fatigue (e.g. EMD_E ~32 vs. 21 ms, EMD_V ~50 vs. 69 ms pre- post-fatigue values, respectively). However, the utility of this mechanism to the prevention of injury may be dependent entirely on routine neuromuscular inhibitory processes (Tsuji and Nakamura, 1988; Zhou et al., 1995; Gleeson, 2001) being down-regulated at critical times of threat to joint stability.

The results of study 3 and 4 offer evidence of the 'direction' of the relationship between fatigue and neuromuscular performance (please see figure 9.1). While the present data showed that acute fatigue could substantively impair the volitional neuromuscular performance capabilities of males and females, there appeared to be a remarkable preservation and indeed potentiation of the capacity of the neuromuscular system following such exercise. Whether these changes to performance capacity reflect a vital mechanism to compensate for reduced

volitional capabilities and preserve dynamic knee joint integrity is currently unclear. In addition, further research is required to investigate how potential inhibitory processes may be manifested during critical periods of joint loading.

Figure 9.1 Locus of anterior cruciate ligament (ACL) injury.



9.1.2.1 *Effects of exercise induced muscle-damage*

The effects of exercise-induced muscle damage (EIMD) on the susceptibility to further injury remains relatively unknown. However consideration of recent pre-season soccer injury data (Hawkins et al., 2001) with the belief that prior physical conditioning can attenuate EIMD symptoms (McHugh et al., 1999), may suggest an increased risk. The eccentric exercise task in study 5 (chapter 8) attempted to challenge the neuromuscular system with a 'worst case' scenario expected of team games by eliciting EIMD in the knee flexors. An acute fatigue task of a similar

nature to that utilised in study 3 was also included to investigate the potential interactive effects of fatigue and EIMD on neuromuscular performance. Commensurate with previous research (Rinard et al., 2000; Nokasa et al., 2001), the eccentric exercise was associated with substantive and prolonged impairments to volitional contractile performance capabilities in addition to an increase in perceived soreness and plasma creatine kinase, that was most prominent at 48 hours following exercise. These data strongly suggest that the eccentric exercise task induced damage in the knee flexors. Despite such changes to the force generating capabilities, there was no significant impairment to EMD_V performance throughout the seven day assessment period. Such observations may offer evidence of a possible compensatory mechanism to conserve temporal neuromuscular performance at vulnerable joint angles. In addition, the magnetically evoked responses indicate a remarkable preservation of the capacity of the neuromuscular system. Given that magnetic stimulation activates the fastest motor units (Maertens de Noordhout, 1991), the maintenance of twitch contractile and temporal responses suggests further that, even during eccentric muscle actions, volitional access to the total capacity of motor units of the knee flexors may be inhibited. A corollary of this interpretations may be that a pool of the fastest, most powerful motor units are maintained in 'reserve' for when true emergency scenarios are perceived.

The peak impairments to the force generating capabilities (at 48 hours) following EIMD were far in excess of the average impairments to these indices of performance subsequent to an acute fatigue task (decreases: PF_V 37.5% vs. 10.1%, RFD_V 65.3% vs. 36.7% following EIMD and fatigue, respectively). In addition, the recovery from pain, a symptom that may cue reduced activity levels, occurred more

rapidly than the restoration of volitional neuromuscular performance. These data may suggest that exercise leading to muscle damage may have greater relative impact on knee injury avoidance capabilities compared to acute muscle fatigue. However, there were no changes to neuromuscular capacity as measured by magnetic stimulation following EIMD and, the acute fatigue task actually induced improvements in EMD_E performance of similar amounts in muscle both asymptomatic and symptomatic of damage (average 15.7% vs. 13.0% improvement, respectively). A possible interpretation of these data may be that in true emergency scenarios, fatigued muscle, even when symptomatic of damage, may possess the potential to access a greater proportion of the capacity of the neuromuscular system when a threat to joint stability is perceived. These processes may compensate for decreased volitional performance levels and restore dynamic knee joint protective capabilities. Such temporal neuromuscular responses to acute fatigue may be an important consideration for the EIMD-symptomatic individual in warm-up settings prior to match-play. However, since EMD_V changes were not observed following fatigue in males in study 3, this research should be repeated with a female sub-sample to investigate the pattern of change to temporal and contractile capabilities in order to better estimate the potential capability to avoid knee injuries in women.

Results for the antagonist muscle group (knee extensors) indicate a minor decrease in RFD_V capabilities of up to 18% at 48 hours. These modifications to knee extensor performance may be influenced consciously or sub-consciously by the sensation of pain in the agonist, or indeed reflect the effects of centrally mediated modification to performance capabilities (Leger and Milner, 2001). Given the

greater absolute performance levels of the knee extensors by comparison to the knee flexors, it is conceivable that such changes may be representative of an attempt to minimise potential muscle group imbalances and the associated potential for increased ACL strain.

9.2 Limitations

9.2.1 *Sample size*

The sample size of study 5 (chapter 8) was limited (n = 7) by comparison to the previous investigations in this thesis. This was due in part to the considerable time commitment required of each individual within this study (> 20 hours). Various aspects of the data including an inability to verify normality of performance responses in such a small sample and unpredictable heterogeneity of response to the EIMD protocol may have ordinarily warranted the deployment of non-parametric methods of data analysis. However, study 5 represented an exploratory investigation, examining aspects of neuromuscular performance not previously considered following EIMD. As such, the application of parametric statistics with their robustness to minor violations of assumptions underpinning their use, seemed reasonable as they were likely to have afforded greater experimental power to detect subtle changes in performance compared to the equivalent non-parametric techniques. Ultimately, this may have helped to provide an indication of whether or not more detailed prospective research might be warranted in the future.

9.2.2 *Testing conditions*

Throughout this thesis, emphasis has been placed on the temporal capability of the individual to muster meaningful levels of muscle force to provide adequate dynamic protection to the knee during critical periods of joint loading. Such capabilities have currently been estimated by means of static MVMAs within a laboratory setting. While these experimental conditions enabled control of the testing environment and minimisation of associated performance variability, they were not representative of a true 'emergency scenario'. Consistently superior EMD_E scores by comparison to EMD_V values, highlight that inhibitory processes may restrict routine volitional access to the total motor unit capacity of the knee musculature. Whether or not such inhibition can be 'down-regulated' may be dependent on the perception of threat to the joint system. Accordingly, future investigations attempting to replicate such scenarios within assessment settings may obtain a truer estimation of the emergency response and knee injury avoidance capabilities.

9.3 Recommendations for future study

The review of literature identified several areas of research which, although not directly addressed in this thesis, should be examined in future work to elucidate further the understanding of the potential relationship between neuromuscular performance, specifically EMD, and ACL injury risk. This relationship should be examined by appropriate prospective randomised trials, similar to those conducted for the index of proprioception (Caraffa et al., 1996; Wedderkopp et al., 1999; Soderman et al., 2000). Prospective trials will help to explore whether or not a causative relationship may exist between EMD and ACL injury, particularly if such

incorporate specific neuromuscular conditioning protocols and examine the adaptive responses of the knee musculature, alongside monitoring incidence of ACL injury. In addition, examination of indices EMD_E and EMD_V during conditions of injury and rehabilitation may reveal whether or not a greater relative difference between such performance capabilities is implicated in ACL injury risk. The results of study 5, which show that the neuromuscular performance capabilities of the knee extensor muscle group may be affected by exercise in the knee flexor group, suggests that the performance capabilities of the antagonist should also be further explored. It is recognised, however, that these types of prospective investigations would require large study population numbers to realise the research aims.

More research is required to examine the mechanisms by which mechanical and metabolic stresses may contribute to neuromuscular performance impairments, which may be associated with knee injury. Such research may consider more 'functionally-relevant' fatiguing protocols involving muscle activation patterns associated with team sports. In addition, it has emerged from this thesis that the temporal capability measured during volitional muscle actions is consistently inferior compared to the capacity observed subsequent to magnetic stimulation. Furthermore this magnetically evoked temporal capacity is, at the very least, preserved following maximal intensity exercise. Future research examining the possible effects of neuromuscular inhibitory mechanisms (Tsuji and Nakamura, 1988; Zhou et al., 1995; Gleeson, 2001) and likely knee injury avoidance capabilities following exercise, may consider investigating such temporal volitional responses during perceived threats to the joint system.

Glossary

GLOSSARY

ACL:	Anterior cruciate ligament.
ANOVA:	Univariate analysis of variance.
CMAP:	Compound muscle action potential. Muscle eletromyographic response subsequent to magnetic stimulation of the associated motor nerve.
CMCT:	Central motor conduction time. Time associated with the propagation of the action potential from the brain to the target site on the spinal cord.
EIMD:	Exercise-induce muscle damage.
EMD_E:	Index of electromechanical delay subsequent to supramaximal magnetically evoked muscle twitch. It is defined as the time delay between the onset of muscle electrical activity and the onset of tension development in skeletal muscle (Norman and Komi, 1979; Zhou et al., 1996).
EMD_V:	Index of electromechanical delay subsequent to maximal voluntary muscle activation.
ES:	Electrical stimulation.
GG:	Greenhouse-Geisser adjustment of the critical F-value where the assumptions underpinning the use of repeated measures ANOVA were violated.
Kinanthropometry:	A scientific discipline which is concerned with the relation between structure and function of the human body, particularly within the context of movement (Eston and Reilly, 2001). Its purpose is also the construction and improvement of measurement techniques to study inter-human variation.
Limb preference:	Refers to the limb with which a subject would prefer to undertake common tasks involving motor control. The preferred limb was identified in this thesis as the limb chosen by the participant to kick a soccer ball with maximum force (e.g. Gleeson et al., 1998b)
MS	Magnetic stimulation.
MVMA	Maximal voluntary muscle activation
p:	Attained significance level.
PMCT	Peripheral motor conduction time. Time associated with the propagation of the action potential from the spinal cord to the target musculature.

PF_V:	Index of static peak force subsequent to maximal voluntary muscle activation where the highest force was recorded.
P_TF_E:	Index of static peak twitch force subsequent to supramaximal magnetically evoked muscle activation where the highest force was recorded.
Reliability:	The statistical reliability associated with an index of neuromuscular performance. Within a given measurement environment, suitable reliability characteristics associated with a given index of neuromuscular performance will ensure that the index is likely to be able to rank the performance capability of members of a group (Gleeson and Mercer, 1992).
Reproducibility:	The relative consistency with which an index of neuromuscular performance is measured during repeated intra-subject testing. Within a given measurement environment, suitable reproducibility characteristics associated with a given index of neuromuscular performance will ensure that the index is likely to be able to detect relatively small changes in performance capability (Gleeson and Mercer, 1992).
R_I:	Intraclass correlation coefficient. This statistical index describes single-measurement reliability (Winer, 1981).
RFD_E:	Index of rate of force development subsequent to supramaximal magnetically evoked muscle twitch, calculated as the average rate of force increase associated with the force-time response between 25 percent and 75 percent of static peak twitch force.
RFD_V:	Index of rate of force development subsequent to maximal voluntary muscle activation, calculated as the average rate of force increase associated with the force-time response between 25 percent and 75 percent of volitional static peak force.
SD:	Standard deviation.
SEC:	Series elastic component. Elastic component in series with the contractile component, comprising predominately of the tendon and connective tissue attachments at the end of the muscle fibres (McComas, 1996), which stretches when the contractile component shortens.

SEM%:	The standard error of a single measurement (computed as a percentage of the group mean score at 95% confidence limits).
Sensitivity:	The ability of an index of performance to detect with certainty relatively small changes in performance capacity (Gleeson and Mercer, 1992).
Supramaximal stimulation:	A plateauing of the amplitude of the muscle compound muscle action potential despite increasing intensities of magnetic stimulation. The beginning of the plateau of the CMAP was defined as the intensity at which no more than a 5% increase in CMAP peak amplitude was observed despite a 10% increases in the intensity of stimulation. This was verified by contemporaneous visual inspection of the data.
$T_{½E}$:	Index of time to half peak twitch force, calculated as the time interval in milliseconds between the observed development of muscle force and the point in time where 50 percent of magnetically evoked static peak twitch force was achieved.
$T_{½V}$:	Index of time to half peak force, calculated as the time interval in milliseconds between the observed development of muscle force and the point in time where 50 percent of volitional static peak force was achieved.
Utility (kinanthropometric):	The logistical ease with which an index can attain suitable reproducibility or reliability and concomitant sensitivity characteristics may be described as a component of its measurement utility (Gleeson and Mercer, 1992, 1996).
V%:	Coefficient of variation.

List of References

LIST OF REFERENCES

Arendt, E.A. and Dick, R. (1995). Knee injury patterns among men and women in collegiate basketball and soccer. *American Journal of Sports Medicine*, **23** (6): 694-701.

Armstrong, R.B., Oglevie, R.W. and Schwane, J.A. (1983). Eccentric exercise-induced injury to rat skeletal muscle. *Journal of Applied Physiology*, **54** (1): 80-93.

Arvidsson, I., Eriksson, E., Haggmark, T. and Johnson, R.J. (1981). Isokinetic thigh muscle strength after ligament reconstruction in the knee joint: results from a 5-10 year follow-up after reconstructions of the anterior cruciate ligament. *International Journal of Sports Medicine*, **2** (1): 7-11.

Aune, A.K., Schaff, P. and Nordsletten, L. (1995). Contraction of the knee flexors and extensors in skiing related to the backward fall mechanism of injury to the anterior cruciate ligament. *Scandinavian Journal of Medicine and Science in Sports*, **5** (2): 165-169.

Barker, A.T., Freeston, I.L., Jalinous, R. and Jarratt, J.A. (1987). Magnetic stimulation of the human brain and peripheral nervous system: an introduction and the results of an initial clinical evaluation. *Neurosurgery*, **20** (1): 100-109.

Balog, E.M. and Fitts, R.H. (1996). Effects of fatiguing stimulation on intramuscular Na^+ and K^+ in frog skeletal muscle. *Journal of Applied Physiology*, **81** (2): 679-685.

Beard, D.J., Kyberd, P.J., Fergusson, C.M. and Dodd, C.A.F. (1993). Proprioception after rupture of the anterior cruciate ligament. *Journal of Bone and Joint Surgery*, **75-B**: 311-315.

Bangsbo, J. (1994). The physiology of soccer. *Acta Physiologica Scandinavia*, **151** (S619)

Behm, D.G. and Sale, D.G. (1994). Voluntary and evoked muscle contractile characteristics in active men and women. *Canadian Journal of Applied Physiology*, **19** (3): 253-265.

Behm, D.G. and St-Pierre, D.M.M. (1997). Effects of fatigue duration and muscle type on voluntary and evoked contractile properties. *Journal of Applied Physiology*, **82** (5): 1654-1661.

Bell, D.G. and Jacobs, I. (1986). Electro-mechanical response times and rate of force development in males and females. *Medicine and Science in Sports and Exercise*, **18** (1): 31-36.

Benecke, R. (1996). Magnetic stimulation in the assessment of peripheral nerve disorders. *Bailliere's Clinical Neurology*, **5** (1): 115-128.

Beynnon, B.D. and Johnson, R.J. (1996). Anterior cruciate ligament injury rehabilitation in athletes. *Sports Medicine*, **22** (1): 54-64.

Biedert, R.M. (2000). Contribution of the three levels of nervous system motor control; spinal cord, lower brain, cerebral cortex. In Lephart, S.M. and Fu, F.H. (eds.), *Proprioception and Neuromuscular Control in Joint Stability*. Champaign Illinois USA: Human Kinetics: 23-30.

Bilcheck, H.M., Kraemer, W.J., Maresh, C.M. and Zito, M.A. (1993). The effects of isokinetic fatigue on recovery of maximal isokinetic concentric and eccentric strength in women. *Journal of Strength and Conditioning Research*, **7** (1): 43-50.

Bochdansky, T., Kollmitzer, J. and Ebinbechler, G. (2000). The Role of Electromyography in the Assessment of Neuromuscular Control. In Lephart, S.M. and Fu, F.H. (eds.), *Proprioception and Neuromuscular Control in Joint Stability*. Champaign Illinois USA: Human Kinetics: 145-159.

Brebner, J.T. and Welford, A.T. (1980). Introduction: an Historical Background Sketch. In Welford, A.T. (eds.), *Reaction Times*. New York USA: Academic Press: 145-159.

Britton, T.C., Meyer, B., Herdmann, J. and Benecke, R. (1990). Clinical use of the magnetic stimulator in the investigation of peripheral conduction time. *Muscle and Nerve*, **13** (3): 396-406.

Brockett, C.L., Morgan, D.L and Proske, U. (2001). Human hamstring muscles adapt to eccentric exercise by changing optimum length. *Medicine and Science in Sports and Exercise*, **33** (4): 783-790.

Brockett, C.L., Morgan, D.L, Gregory, J.E. and Proske, U. (2002). Damage to different types of motor units following repeated active lengthenings of the medial gastrocnemius muscle of the cat. *Journal of Applied Physiology*, **92** (3): 1104-1110.

Brown, S.J., Child, R.B., Day, S.H. and Donnelly, A.E. (1997). Exercise-induced skeletal muscle damage and adaptation following repeated bouts of eccentric muscle contractions. *Journal of Sports Sciences*, **15** (2): 215-222.

Brown, S.J., Child, R.B., Donnelly, A.E., Saxton, J.M. and Day, S.H. (1996). Changes in human skeletal muscle contractile function following stimulated eccentric exercise. *European Journal of Applied Physiology*, **72** (5-6): 515-521.

Bolgla, L.A. and Keskula, D.R. (2000). A review of the relationship among knee effusion, quadriceps inhibition, and knee function. *Journal of Sport Rehabilitation*, **9** (2): 160-168.

Bollen, S. (1998). Ligament injuries of the knee - limping forward. British *Journal of Sports Medicine*, **32** (1): 82-84.

Byrne, C. and Eston, R.G. (1998). Exercise, muscle damage and delayed onset muscle soreness. *Sports Exercise and Injury*, **4** (1): 67-70.

Byrne, (2001). Changes of isometric and dynamic strength loss following exercise-induced muscle damage. *Scandinavian Journal of Medicine and Science in Sports and Exercise*, **11** (2): 134-140.

Caraffa, A., Cerulli, G., Projetti, M., Aisa, G. and Rizzo, A. (1996). Prevention of anterior cruciate ligament injuries in soccer. *Knee Surgery, Sports Traumatology, Arthroscopy*, **4** (1): 19-21.

Cavanagh, P.R. and Komi, P.V. (1979). Electromechanical delay in human skeletal muscle under concentric and eccentric contractions. *European Journal of Applied Physiology and Occupational Physiology*, **42** (2): 159-163.

Chan, A.Y.F., Lee, F.L.L., Wong, P.K., Wong, C.Y.M and Yeung, S.S. (2001). Effects of knee joint angles and fatigue on the neuromuscular control of vastus medialis oblique and vastus laterals muscle in humans. *European Journal of Applied Physiology*, **84** (1): 36-41.

Chokroverty, S., Flynn, D., Picone, M.A., Chokroverty, M. and Belsh, J. (1993). Magnetic coil stimulation of the human lumbosacral vertebral column: site of stimulation and clinical application. *Electroencephalography and Clinical Neurophysiology*, **89** (1): 54-60.

Cross, D., Chiappa, K.H., Gominak, S., Fang, J., Santamaria, J., King, P.J. and Shahani, B.T. (1990). Cervical magnetic stimulation. *Neurology*, **40** (11): 1751-1756.

Currier, D.P. (1984). *Elements of Research in Physical Therapy* (2nd Edition.). USA, Baltimore Williams and Wilkins.

Deie, M., Sakamaki, Y., Urabe, Y. and Ikuta, Y (2002). Anterior knee laxity in young women varies with their menstrual cycle. *International Orthopaedics*, **26** (3): 154-156.

De Luca, C.J. (1997). The use of surface electromyography in biomechanics. *Journal of Applied Biomechanics*, **13** (2): 135-163

Devita, P. And Skelly, W.A. (1992). Effect of landing stiffness on joint kinetics and energetics in the lower extremity. *Medicine and Science in Sports and Exercise*, **24** (1): 108-115.

van Dieen, J.H., Thissen, C.E.A.M., van de Ven, A.J.G.M. and Toussaint, H.M. (1991) The electromechanical delay of the errector spinae muscle: influence of

electrode location. *European Journal of Applied Physiology and Occupational Physiology*, **63** (3): 216-222.

Dilitto, A., Rose, S.J., McKowen, J.M., Lehman, R.C., Thomas, J.A. and Shilvely, R.A. (1988). Electrical stimulation versus voluntary exercise strengthening thigh musculature after anterior cruciate ligament surgery. *Physical Therapy*, **68** (5): 660-663.

Enoka, R.M. and Fuglevand, A.J. (2001). Motor unit physiology: some unresolved issues. *Muscle Nerve*, **24** (1): 4-17.

Eston, R.G. and Reilly, T. (eds.), *Kinanthropometry and Exercise Physiology Laboratory Manual: Tests, Procedures and Data* (2nd Edition). London: Routledge

Evans, B.A., Litchy, W.J. and Daube, J.R. (1988). The utility of magnetic stimulation for routine peripheral nerve conduction studies. *Muscle and Nerve*, **11** (10): 1074-1078.

Ferris-Hood, K., Threlkeld, A.J., Horn, T.S. and Shapiro, R.A. (1996). Electromechanical delay of the quadriceps during selected movement velocities. *Electromyography and Clinical Kinesiology*, **36** (3) 157-170.

Fitts, R.H. (1996). Muscle fatigue: the cellular aspects. *American Journal of Sports Medicine*, **24** (6): s9-s13.

Foley, J.M., Jayraman, R.C., Prior, B.M., Pivarnik, J.M. and Meyer, R.A. (1999). MR measurements of muscle damage and adaptation after eccentric exercise. *Journal of Applied Physiology*, **87** (6): 2311-2318.

Friden, J. and Lieber, R.L. (1992). Structural and mechanical basis of exercise-induced muscle injury. *Medicine and Science in Sports and Exercise*, **24** (5): 521-530.

Fu, F.H., Harner, C.D., Johnson, D.L., Miller, M.D. and Woo, S.L-Y. (1993). Biomechanics of knee ligaments. *Journal of Bone and Joint Surgery*, **75-A** (11): 1716-1727.

Gabriel, D.A. and Boucher, J.P. (1998). Effects of repetitive dynamic contractions upon electromechanical delay. *European Journal of Applied Physiology*, **79** (1): 37-40.

Garnett, R. and Stephens, J.A. (1981). Changes in the recruitment threshold of motor units produced by cutaneous stimulation in man. *Journal of Physiology*, **311** (1): 463-473.

Gleeson, M., Blannin, A.K., Zhu, B., Brookes, S. and Cave, R. (1995). Cardiorespiratory, ormonal and haematological responses to submaximal cycling performed 2 days after eccentric or concentric exercise bouts. *Journal of Sports Sciences*, **13** (6): 471-479.

Gleeson, N.P. (2001). Assessment of neuromuscular performance using electromyography. In Eston, R.G. and Reilly, T. (eds.), *Kinanthropometry and Exercise Physiology Laboratory Manual: Tests, Procedures and Data* (2nd Edition). London: Routledge: 37-63.

Gleeson, N.P., Eston, R., Marginson, V. and McHugh, M. (2003). Effects of prior concentric training on eccentric exercise-induced muscle damage. *British Journal of Sports Medicine,* **37** (2): 119-125

Gleeson, N.P. and Mercer, T.H. (1992). Reproducibility of isokinetic leg strength and endurance characteristics of adult men and women. *European Journal of Applied Physiology and Occupational Physiology*, **65** (3): 221-228.

Gleeson, N.P. and Mercer, T.H. (1996). The utility of isokinetic dynamometry in the assessment of human muscle function. *Sports Medicine*, **21** (1): 18-34.

Gleeson, N.P., Mercer, T.H., Morris, K. and Rees, D. (1997). Influence of a fatigue task on electromechanical delay in the knee flexors of soccer players. *Medicine and Science in Sports and Exercise*, **29** (5): S281.

Gleeson, N.P., Naish, P.F., Mercer, T.H., Wilcock, J.E. and Hine, J. (2002). Reliability of indices of leg neuromuscular performance in end-stage renal failure. *Scandinavian Journal of Rehabilitation Medicine*, **34** (6): 273-277.

Gleeson, N.P., Rees, D., Doyle, J.M., Minshull, C., Walters, M. and Bailey, A. (1998a). Effects of anterior cruciate ligament reconstructive surgery and acute physical rehabilitation on neuromuscular modelling associated with knee the joint. *British Association of Sport and Exercise Sciences Annual Conference*, Worcester College, November.

Gleeson, N.P., Rees, D., Glover, D., Minshull, C. and Walters, M. (2000). Effects of a fatigue task on the neuromuscular performance in the knee flexors of high-performance soccer players. In: Avela, J., Komi, P.V. and Komulainen, J. (eds.). *Proceedings of the 5th Annual Congress of the European College of Sport Science*, Jyvaskula, Finland, July: LIKES Research Centre: 287ff.

Gleeson, N.P., Rees, D., Rakowski, S. and Reilly, T. (1995). Reproducibility of indices of anterior tibio-femoral displacement in active and inactive men. In: Atkinson, G. and Reilly, T. (eds.) *Sport Leisure and Ergonomics*. London: E and FN Spon: 198-203.

Gleeson, N.P., Reilly, T., Mercer, T.H., Rakowski, S., Rees, D. (1998b). Influence of acute endurance activity on leg neuromuscular and musculoskeletal performance. *Medicine and Science in Sports and Exercise*, **30** (4): 596-608.

Granata, K.P., Ikeda, A.J. and Abel, M.F. (2000). Electromechanical delay and reflex response in spastic cerebral palsy. *Archives of Medical Rehabilitation*, **81** (7): 888-894.

Grabiner, M.D. (1986). Bioelectrical characteristics of the electromechanical delay preceding concentric contraction. *Medicine and Science in Sports and Exercise*, **18** (1): 37-43.

Gray, J., Taunton, J.E., McKenzie, D.C., Clement, D.B., McConkey, J.P. and Davidson, R.G. (1985). A survey of injuries to the anterior cruciate ligament of the knee in female basketball players. *International Journal of Sports Medicine*, **6** (6): 314-316.

Hakkinen, K. and Komi, P.V. (1983). Changes in neuromuscular performance in voluntary and reflex contraction during strength training. *International Journal of Sports Medicine*, **4** (4): 282-288.

Hamada, T., Sale, D.G., MacDougal, J.D. and Tarnopolsky, M.A. (2000). Postactivation potentiation, fibre type and twitch contraction time in human knee extensor muscles. *Journal of Applied Physiology*, **88** (6): 2131-2137

Harmon, K.G and Ireland, M.L. (2000). Gender differences in noncontact anterior cruciate ligament injuries. *Clinics in Sports Medicine*, **19** (2): 287-302.

Hawkins, R., Hulse, M. and Hodson, A. (2001). An audit of injuries in professional football. *The Football Association*, Newport, Shropshire.

Hirshman, P.H., Daniel, D.M. and Miyasaka, K. (1990). The fate of unoperated knee ligament injuries. In Daniel, D., Akeson, W. and O'Connor, J (eds.), *Knee*

Ligaments: Structure, Function, Injury and Repair. New York USA: Raven Press: 481-503.

Hollge, J., Kunkel, M., Ziemann, U., Tergau, F., Geese, R. Reimers, C.D. (1997). Central fatigue in sports and daily exercise a magnetic stimulation study. *International Journal of Sports Medicine*, **18** (8): 614-617.

Holloway, J.B. and Baechle, T. (1990). Strength training for female athletes - a review of selected aspects. *Sports Medicine*, **9** (4): 216-228.

Hopkins, W.G. (2000). Measures of reliability in sports medicine and science. *Sports Medicine*, **30** (1): 1-15.

Hopkins, J.T. and Ingersoll, C.D. (2000). Arthrogenic muscle inhibition: a limiting factor in joint rehabilitation. *Journal of Sport Rehabilitation*, **9** (2): 135-159.

Horita, T. and Ishiko, T. (1987). Relationships between muscle lactate accumulation and surface EMG activities during isokinetic contractions in man. *European Journal of Applied Physiology and Occupational Physiology*, **56** (1): 18-23.

Hortobagyi, T., Houmard, J., Fraser, D., Dudek, R., Lambert, J. and Tracy, J. (1998). Normal forces and myofibrillar disruption after repeated eccentric exercise. *Journal of Applied Physiology*, **82** (2): 492-498.

Houston, M.F., Norman, R.W. and Froese, E.A. (1988). Mechanical measures during maximal velocity knee extension exercise and their relation to fibre composition of the human vastus lateralis muscle. *European Journal of Applied Physiology and Occupational Physiology*, **58** (1): 1-7.

Hutchinson, M.R. and Ireland, M.L. (1995). Knee injuries in female athletes. *Sports Medicine*, **19** (4): 288-302.

Ingalls, C.P., Warren, G.L., Williams, J.H., Ward, C.W. and Armstrong, R.B. (1998). E-C coupling failure in mouse EDL muscle after in vivo eccentric contractions. *Journal of Applied Physiology*, **85** (1): 58-67.

Ireland, M.J., Gaudette, M. and Crook, S. (1997). ACL injuries in the female athlete. *Journal of Sport Rehabilitation*, **6** (2): 97-110.

Ireland, M.J. and Wall, C. (1990). Epidemiology and comparison of knee injuries in elite male and female United States basketball athletes. *Medicine and Science in Sports and Exercise*, **22** (S82).

Jalinous, R. (1995). *Guide to Magnetic Stimulation.* The Magstim Company, Whitland, Wales UK.

Johansson, H. (1991). Role of knee ligaments in proprioception and regulation of muscle stiffness. *Journal of Electromyography and Clinical Neurophysiology*, **1** (3): 158-179.

Johansson, H., Sjolander, P. and Sojka, P. (1991). A sensory role for the cruciate ligaments. *Clinical Orthopaedics and Related Research*, **262**: 161-178.

Jones, D.A., Newham, D.J. and Torgan, C. (1989). Mechanical influences on long-lasting human muscle fatigue and delayed-onset pain. *Journal of Physiology*, **412** (1): 415-427.

Kaneko, F., Onari, K., Kawaguchi, K., Tsukisaka, K. and Roy, S.H. (2002). Electromechanical delay after ACL reconstruction: an innovative method for investigating central and peripheral contributions. *Journal of Orthopaedic and Sports Physical Therapy*, **32** (4): 158-165.

Kent-Braun, J.A. (1999). Central and peripheral contributions to muscular effort to muscle fatigue in humans during sustained maximal effort. *European Journal of Applied Physiology*, **80** (1): 57-63.

King P.J.L. and Chippa, K.H. (1989). Motor Evoked Potentials, In; Chippa (ed). *Evoked Potentials in Clinical Medicine*. USA: Raven Press.

Klimovitch, G. (1977). Startle response and muscular fatigue effects upon fractionated hand grip reaction time. *Journal of Motor Behaviour*, **9** (4): 285-292.

Komi, P.V. (1979). Neuromuscular performance: factors influencing force and speed production. *Scandinavian Journal of Sports Science*, **1** (1): 2-15.

Kubo, K., Akima, H., Kouzaki, M., Ito, M., Kawakami, Y., Kanehisa H. and Fukunaga, T. (2000). Changes in the elastic properties of tendon structures following 20 days of bed-rest in humans. *European Journal of Applied Physiology*, **83** (6): 463-468.

Kujala, U.M., Friberg, O., Aalto, T. And Osterman, K. (1987). Lower limb asymmetry and patellofemoral joint incongruence in the etiology of knee exertion injuries in athletes. *International Journal of Sports Medicine*, **8** (3): 214-220.

Kyrolainen, H. and Komi, P.V. (1994). Neuromuscular performance of lower limbs during voluntary and reflex activity in power- and endurance-trained athletes. *European Journal of Applied Physiology and Occupational Physiology*, **69** (3): 233-239.

Latash, M.L. (1998). *Neurophysiological Basis of Movement*. Champaign, Ill. USA: Human Kinetics.

Lattanzio, P-J., Petrella, R.J., Sproule, J.R. and Fowler, P.J. (1997). Effects of fatigue on knee proprioception. *Clinical Journal of Sport Medicine*, **7** (1): 22-27.

Linnamo, V., Bottas, R. and Komi, P.V. (2000). Force and EMG power spectrum during and after eccentric and concentric fatigue. *Journal of Electromyography and Kinesiology*, **10** (5): 293-300.

Lipsey, M.W. (1990). *Design Sensitivity: Statistical Power for Experimental Research.* London, England: SAGE Publications.

Leger, A.B. and Milner, T.E. (2000). Muscle function at the wrist after eccentric exercise. *Medicine and Science in Sports and Exercise*, **33** (4): 612-620.

Maertens de Noordhout, A. (1991). *Percutaneous magnetic stimulation of central and peripheral pathways*. Course proceedings of the Belgian Society of Electromyography and Clinical Neurophysiology, Brussels Foundation Univeversitaire, March.

Marginson, V. (2003). *Symptoms of Exercised-Induced Muscle Damage in Boys and Men Following Eccentric Exercise*. Unpublished Ph.D. thesis, University of Wales, Bangor.

McComas, A.J. (1996). *Skeletal Muscle: Form and Function*, Champaign, Illinois: Human Kinetics.

McHugh, M., Connolly, D.A.J., Eston, R.G., Kremenic, I.J., Nicholas, S.J. and Gleim, G.W. (1999). The role of passive muscle stiffness in symptoms of exercise-induced muscle damage. *American Journal of Sports Medicine*, **27** (5): 594-599.

McHugh, M., Connolly, D.A.J., Eston, R.G. and Gleim, G.W. (2000). Electromyographic analysis of exercise resulting in symptoms of muscle damage. *Journal of Sports Sciences*, **18** (3) 163-172.

McInnes, S.E., Carlson, J.S., Jones, C.J. and McKenna, M.J. (1995). The physiological load imposed on basketball players during competition. *Journal of Sports Sciences*, **13** (5): 387-397.

Mercer, T.H. and Gleeson, N.P. (2002). The efficacy of measurement and evaluation in evidence-based clinical practice. *Physical Therapy in Sport*, **3** (1): 1-9.

Mercer, T.H., Gleeson, N.P., Claridge, S. and Clement, S. (1988). Prolonged intermittent high intensity exercise impairs neuromuscular performance of the knee flexors. *European Journal of Applied Physiology and Occupational Physiology*, **77** (6): 560-562.

Metzger, J.M and Fitts, R.H. (1987). Role of intracellular pH in muscle fatigue. *Journal of Applied Physiology*, **62** (4): 1392-1397.

Moore, M.A and Kukulka, C.G. (1991). Depression of hoffman reflexes following voluntary contraction and implications for proprioceptive neuromuscular facilitation therapy. *Physical Therapy*, **71** (4): 69-77.

Morgan, D.L. and Allen D.G. (1999). Early events in stretch-induced muscle damage. *Journal of Applied Physiology*, **87** (6): 2007-2015.

Morris, A.F. and Beaudet, S.M. (1980). Electromyographic latencies associated with rapid maximal force production in five different muscle groups in college adults. *American Correctional Therapy Journal*, **34** (4): 116-121.

Morrissey, M.C., Brewster, C.E., Shields, C.L. and Brown, M. (1985). The effects of electrical stimulation on the quadriceps during postoperative knee immobilisation. *American Journal of Sports Medicine*, **13** (1): 40-45.

Murray, N.M.F. (1991). Magnetic stimulation of cortex: clinical applications. *Journal of Clinical Neurophysiology*, **8** (1): 66-76.

Nokasa, K., Sakamoto, K., Newton, M. and Sacco, P. (2001). How long does the protective effect on eccentric exercise-induced muscle damage last? *Medicine and Science in Sport and Exercise*, **33** (9): 1490-1495.

Norman, R.W. and Komi, P.V. (1979). Electromechanical delay in skeletal muscle under normal movement conditions. *Acta Physiologica Scandinavica*, **106** (4): 241-248.

Noyes, F.R., Mooar, L.A., Moorman, C.T. and McGinniss, G.H. (1989). Partial tears of the anterior cruciate ligament. *Journal of Bone and Joint Surgery*, **71-B** (5): 825-833.

Noyes, F.R., Mooar, L.A., Matthews, D.S. and Butler, D.L. (1983). The symptomatic anterior cruciate-deficient knee: the long term functional disability in athletically active individuals. *Journal of Bone and Joint Surgery*, **65-A** (2): 154-174.

O'Leary, D.D., Hope, K. and Sale, D.G. (1997). Posttetanic potentiation of human dorsiflexors. *Journal of Applied Physiology*, **83** (6): 2131-2138.

Paasuke, M., Ereline, J. and Gapeyeva, H. (1999). Neuromuscular fatigue during repeated exhaustive submaximal static contractions of knee extensor muscles in endurance-trained, power-trained and untrained men. *Acta Physiologica Scandinavica*, **166** (4): 319-326.

Polkey, M.I., Kyroussis, D., Hamnegard, C.H., Mills, G.H., Green, M. and Moxham, J. (1996). Quadriceps strength assessed by magnetic stimulation of the femoral nerve in man. *Muscle and Nerve*, **19** (5): 549-555.

Pope, M.H., Johnson, R.J., Brown, D.W. and Tighe, C (1979). The role of the musculature in injuries to the medial collateral ligament. *Journal of Bone and Joint Surgery*, **61-A** (3): 398-402.

Rassier, D.E. and MacIntosh, B.R. (2000). Coexistance of potentiation and fatigue in skeletal muscle. *Brazilian Journal of Medical and Biological Research*, **33** (5): 499-508.

Rees, D. (1994). *Failed ACL reconstructions*. Proceedings of the Football Association - Royal College of Surgeons, Edinburgh 6th Joint Conference on Sport Injury, Lilleshall Hall National Sports Centre, July.

Rinard, J., Clarkson, P.M., Smith, L.L. and Grossman, M. (2000). Response of males and females to high-force eccentric exercise. *Journal of Sports Sciences*, **18** (4): 229-236.

Romani, W., Patrie, J., Curl, L.A. and Flaws, J.A. (2003). The correlations between estradiol, estrone, estriol, progesterone and sex-binding globin and anterior cruciate ligament stiffness in healthy, active females. *Journal of Womens Health*, **12** (3): 287-298

Roos, H. and Karlsson, J. (1998). Anterior cruciate ligament instability and reconstruction. *Scandinavian Journal of Medicine and Science in Sports*, **8** (6): 426-431.

Sahlin, K. and Ren, J.M. (1989). Relationship of contraction capacity to metabolic changes during recovery from a fatiguing contraction. *Journal of Applied Physiology*, **67** (2): 648-654.

Sahlin, K. and Seger, J.Y. (1995). Effects of prolonged exercise on the contractile properties of human quadriceps muscle. *European Journal of Applied Physiology and Occupational Physiology*, **71** (2-3): 180-186.

Saxton J. M., Clarkson, P.M., James, R., Miles, M., Westerfer, M., Clark, S. and Donnelly, A.E. (1995). Neuromuscular dysfunction following eccentric exercise. *Medicine and Science in Sports and Exercise*, **27** (8): 1185-1193.

Shelbourne, D.K. and Foulk, D.A. (1995). Timing of surgery in acute anterior cruciate ligament tears on the return of quadriceps muscle strength after reconstruction using an autogenous patellar tendon graft. *American Journal of Sports Medicine*, **23** (6): 686-689.

Shelbourne, D.K. and Gray, T. (1997). Anterior cruciate ligament reconstruction with autogenous patellar tendon graft followed by accelerated rehabilitation. *American Journal of Sports Medicine*, **25** (6): 786-795.

Slauterbeck, J.R., Fuzie, S.F., Smith, M.P., Clark, R.J., Starch, D.W and Hardy, D.M. (2002). The menstrual cycle, sex hormones and anterior cruciate ligament injury. *Journal of Athletic Training*, **37** (3): 275-278

Soderman, K., Werner, S., Pietila, T., Engstrom, B. and Alfredson, H. (2000). Balance board training: prevention of traumatic injuries of the lower extremities in female soccer players. *Knee Surgery, Sports Traumatology, Arthroscopy*, **8** (6): 356-363.

Sokal, R. and Rohlf, F. (1981). *Biometry* (2nd Ed), Oxford, UK, W.H. Freeman.

Stone, M.H. (1992). Connective tissue and bone responses to strength training. In: Komi, P.V. (Ed.). *Strength and Power in Sport*. Champaign, IL.: Human Kinetics

Strojnik, V. and Komi, P.V. (1998). Neuromuscular fatigue after maximal stretch-shortening cycle exercise. *Journal of Applied Physiology*, **84** (1): 344-350.

Stupka, N., Tarnopolsky, M.A., Yardley, N.J. and Phillips, S.M. (2001). Cellular adaptation to repeated eccentric exercise-induced muscle damage. *Journal of Applied Physiology*, **91** (4): 1669-1678.

Swanik, B.C., Lephart, S.M., Giannantonio, F.P. and Fu, F.H. (1997). Reestablishing proprioception and neuromuscular control in the ACL-injured athlete. *Journal of Sport Rehabilitation*, **6** (2): 182-206.

Snyder-Mackler, L., Binder-Macleod, S.A. and Williams, P.R. (1993). Fatigability of human quadriceps femoris muscle following anterior cruciate ligament reconstruction. *Medicine and Science in Sports and Exercise*, **25** (7): 783-798.

Snyder-Mackler, L., Delitto, A., Stralka, S.W. and Bailey, S.L. (1994). Use of electrical stimulation to enhance recovery of quadriceps femoris muscle force production in patients following anterior cruciate ligament surgery. *Physical Therapy*, **74** (10): 901-907.

Taylor, D.C., Brooks, D.E. and Ryan, J.B. (1997). Viscoelastic characteristics of muscle: passive stretching versus muscular contractions. *Medicine and Science in Sports and Exercise*, **29** (12): 1619-1624.

Thomas, J.R. and Nelson, J.K. (1996). *Research Methods in Physical Activity*. Champaign Illinois USA: Human Kinetics.

Tsuji, I. and Nakumura, R. (1988). Time course of tension development of knee extensor muscle on twitch, tetanic and fast voluntary contraction in normal subjects. *Tohoku Journal of Experimental Medicine*, **155** (3): 225-232.

Ugawa, Y., Rothwell, J.C., Day, B.L., Thompson, P.D. and Marsden, C.D. (1989). Magnetic stimulation over the spinal enlargements. *Journal of Neurology, Neurosurgery and Psychiatry*, **52**: 1025-1032.

Viitasalo, J.T. and Komi, P.V. (1981). Interrelationships between electromyographical, muscle structure and reflex time measurements in man. *Acta Physiologica Scandinavica*, **111** (1): 97-103.

Viitasalo, J.T., Saukkonen, S. and Komi, P.V. (1980). Reproducibility of measurements of selected neuromuscular performance variables in man. *Electromyography and Clinical Neurophysiology*, **20** (6): 487-501.

Vincent, H.K. and Vincent, K.R. (1997). The effect of training status on the serum creatine kinase response, soreness and muscle function following resistance exercise. *International Journal of Sports Medicine*, **18** (6): 431-437.

Vint, P.F., McLean, S.P. and Harron, G.M. (2000). Electromechanical delay in isometric actions initiated from nonresting levels. *Medicine and Science in Sports and Exercise*, **33** (6): 978-983.

Vos, E.J., Harlaar, J. and van Ingen Schenau, G.J. (1991). Electromechanical delay during knee extensor contractions. *Medicine and Science in Sports and Exercise*, **23** (10): 1187-1193.

Vos, E.J., Mullender, M.G. and van Ingen Schenau, G.J. (1990). Electromechanical delay in the vastus lateralis muscle during dynamic contractions. *European Journal of Applied Physiology and Occupational Physiology*, **60** (6): 467-471.

Warren, G.L., Hermann, K.M., Ingalls, C.P., Massell, M.R. and Armstrong, R.B. (2000). Decreased EMG median frequency during a second bout of eccentric contractions. *Medicine and Science in Sports and Exercise*, **32** (4): 820-829.

Wedderkopp, N., Kaltoft, M., Lundgaard, B., Rosendahl, M. and Froberg, K. (1999). Prevention of injuries in young female handball players on European team handball. A prospective intervention study. *Scandinavian Journal of Medicine and Science in Sports*, **9** (1): 41-47.

West, W., Hicks, A., McKelvie, R. and O'Brien, J. (1996). The relationship between plasma potassium, muscle membrane excitability and force following quadriceps fatigue. *Pflugers Archive European Journal of Physiology*, **432** (1): 43-49.

Williams, K.M. (2002). *Neuromuscular performance and the extent of fatigue in the knee flexors during periods of eccentric exercise-induced muscle damage.* University of Wales, Bangor, unpublished Masters Thesis.

Winer, B.J. (1981). *Statistical principles in experimental design.* (2nd Ed.), New York USA, McGraw-Hill.

Winter, E.W. and Brookes, F.B.C. (1991). Electromechanical response times and muscle elesticity in men and women. *European Journal of Applied Physiology and Occupational Physiology*, **63** (2): 124-128.

Wojtys, E.M., Huston, L.J. (1996). Neuromuscular performance characteristics of elite female athletes. *American Journal of Sports Medicine*, **24** (4): 427-436.

Wojtys, E.M., Huston, L.J., Lindenfeld, T.N, Hewett, T.E. and Greenfield, M.L. (1998). Association between the menstrual cycle and anterior cruciate ligament injuries in female athletes. *American Journal of Sports Medicine*, **26** (5): 614-619.

Yeung, S.S., Au, A. and Chow, C.C. (1999). Effects of fatigue on the temporal neuromuscular control of vastus medialis muscle in humans. *European Journal of Applied Physiology*, **80** (4): 379-385.

Zhou, S, (1996). Acute effect of repeated maximal isometric contraction on electromechanical delay of knee extensor muscle. *Journal of Electromyography and Kinesiology*, **6** (2): 117-127.

Zhou, S., Lawson, D.L., Morrison, W.E. and Fairweather, I. (1995). Electromechanical delay in isometric muscle contractions evoked by voluntary, reflex and electrical stimulation. *European Journal of Applied Physiology and Occupational Physiology*, **70** (2): 138-145.

Zhou, S., McKenna, M., Lawson, D.L., Morrison, W.E. and Fairweather, I. (1996). Effects of sprint training on electromechanical delay of knee extensor muscles. *European Journal of Applied Physiology and Occupational Physiology*, **72** (5-6): 410-416.

Zhou, S., Carey, M.F., Snow, R.J., Lawson, D.L. and Morrison W.E. (1998). Effects of muscle fatigue and temperature on electromechanical delay. *Electromyography and Clinical Neurophysiology*, **38** (2): 68-73.

Zhu, Y., Starr, A., Hwan Su, S., Woodward, G. and Haldeman, S. (1992). The H-reflex to magnetic stimulation of lower-limb nerves. *Archives of Neurology*, **49** (1): 66-71.

Appendices

Appendix A

Example informed consent and medical questionnaire

SCHOOL OF SPORT, HEALTH AND EXERCISE SCIENCES
UNIVERSITY OF WALES, BANGOR

INFORMED CONSENT
& MEDICAL QUESTIONNAIRE

Name: _____

Age: _____

Are you in good health? Yes/No
If no, please explain:

How would you describe your present level of vigorous activity?
Vigorous (please circle):
less than once per month
Once per month
2-3 times per week
4-5 times per week
>5 times per week

Have you ever suffered from a serious illness or accident? Yes/No
If yes, please give particulars:

Do you suffer, or have you ever suffered from:
Asthma Yes/No
Diabetes Yes/No
Bronchitis Yes/No
Epilepsy Yes/No
High blood pressure Yes/No
High cholesterol Yes/No
Are you currently taking medication? Yes/No
If yes, please give particulars:

Are you currently attending your GP for any condition or
have you consulted your doctor in the last three months? Yes/No
If yes, please give particulars:

Have you, or are you presently taking part in any other laboratory experiment? Yes/No
Do you smoke? Yes/No
If you smoke, how many cigarettes do you smoke per day?

If you have smoked previously, approximately how long ago did you stop?

PLEASE READ THE FOLLOWING CAREFULLY

Persons will be considered unfit to do the experimental exercise if they:

Have a fever, suffer from fainting spells or dizziness;
Have suspended training due to joint or muscle injury;
Have a known history of family history of medical disorders, i.e. high blood pressure, high cholesterol, heart or lung disease;
Have had hyper/hypothermia, heat exhaustion, or any other heat or cold disorder;
Have anaphylactic shock symptoms to needles, probes, or other medical-type equipment;
Have chronic or acute symptoms of gastrointestinal bacterial infections (e.g. dysentery, salmonella);
Have a history of infectious diseases (e.g., HIV, Hepatitis B); and if appropriate to the study design, have a known history of rectal bleeding, anal fissures, haemorrhoids, or any other condition of the rectum.

DECLARATION
I hereby volunteer to be a participant in experiments/investigations during the period of
_____2001.

My replies to the above questions are correct to the best of my belief and I understand that they will be treated with the strictest confidence. The experimenter has explained to my satisfaction the purpose of the experiment and possible risks involved.

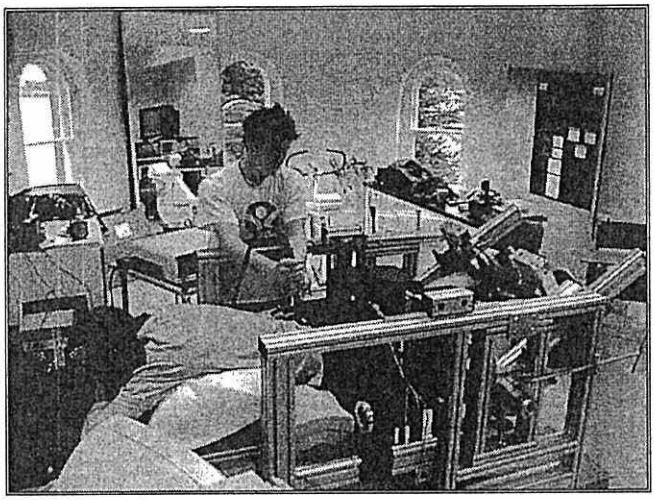
Furthermore, if I am a student I am aware that taking part or not taking part in this experiment, will neither be detrimental to, or further my position as a student.

For my own protection and the protection of others, I undertake to obey the laboratory/study regulations and the instructions of the experimenter regarding the health and safety issues pertaining to this experiment.
I understand I may withdraw from the experiment at any time and that I am under no obligation to give reasons for withdrawal or to attend again for experimentation.

Signature of participant _____
Date _____
Signature of experimenter _____
Date _____

Appendix B

Photographs of selected experimental procedures



Peripheral magnetic stimulation of the sciatic nerve of the right leg.

Image depicts the dynamometer, participant orientation, securing devices and magnetic coil situated in the lumbrosacral region.

Peripheral magnetic stimulation of the sciatic nerve of the right leg.

(Alternative view)

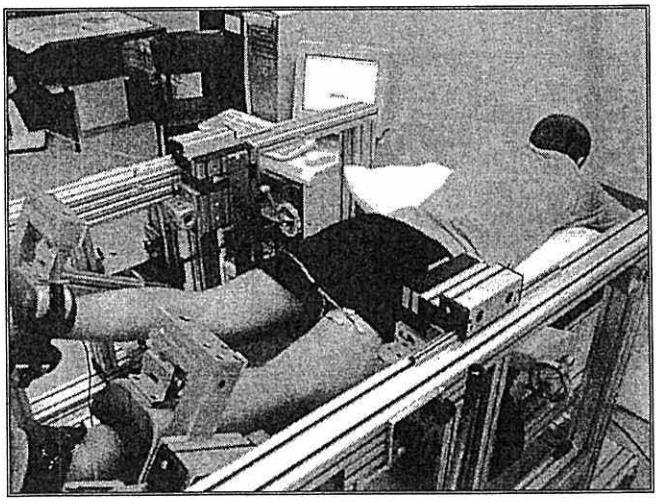
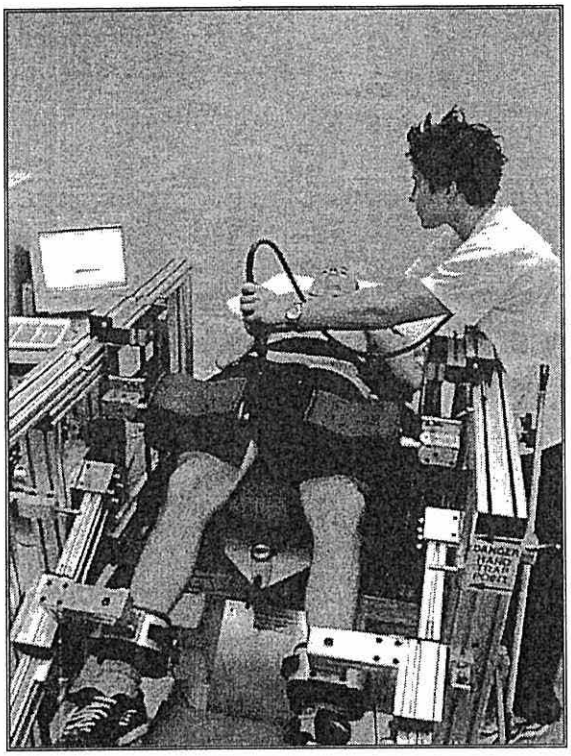


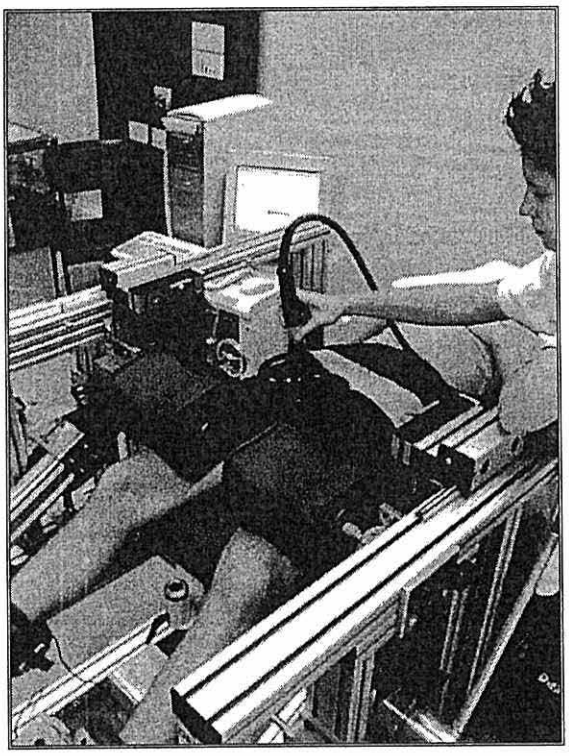
Image showing placement of EMG surface electrodes on the m. biceps femoris of the right leg.

(Posterior knee restraints removed for visual purposes)



Peripheral magnetic stimulation of the femoral nerve of the right leg.

Image depicts the dynamometer, participant orientation, securing devices and magnetic coil situated in the femoral triangle.



Peripheral magnetic stimulation of the femoral nerve of the right leg.

(Alternative view)

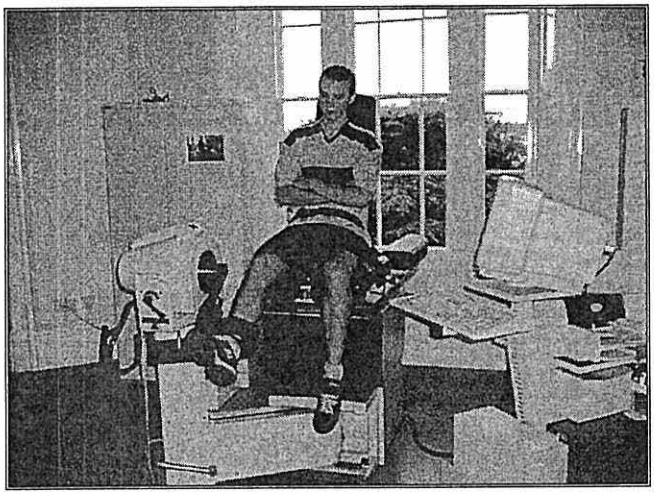


Image showing participant orientation on the isokinetic dynamometer (KinCom) used to elicit exercise-induced muscle damage by means of eccentric activation of the knee flexors (right leg).

Appendix C

Example of supramaximal checking procedure

Example of magnetically evoked responses of the knee flexors of one participant.

Index		40%	50%	60%	70%	80%	70-80% %change	90%	80-90% %change	100%	90-100% %change
AMP	(mV)	0.036	1.56	6.31	6.38	6.23	**-2.4**	6.28	**0.90**	6.64	**5.7**
EMD_E	(ms)	35.1	28.7	27.4	25.9	22.1	**-14.7**	20.0	**-9.5**	20.6	**4.2**
P_TF_E	(N)	6.0	12.5	21.7	26.1	32.1	**23.0**	37.0	**15.3**	40.2	**8.7**

Example of magnetically evoked responses of the knee extensors of one participant.

Index		40%	50%	60%	70%	80%	70-80% %change	90%	80-90% %change	100%	90-100% %change
AMP	(mV)	1.20	4.16	6.99	8.14	8.40	**3.2**	8.50	**1.2**	8.61	**1.3**
EMD_E	(ms)	21.6	18.9	18.8	18.0	16.3	**-8.9**	16.1	**-1.8**	16.9	**4.9**
P_TF_E	(N)	17.0	44.9	69.1	75.1	79.8	**6.3**	76.2	**-4.6**	78.0	**2.4**

The beginning of the plateau of the CMAP was defined as the intensity at which no more than a (~) 5% increase in CMAP peak amplitude was observed despite a 10% increases in the intensity of stimulation. On occasions where supramaximal stimulation was not achieved by this criteria, supplementary tests of peak twitch force (P_TF_E) and electromechanical delay (EMD_E) showed no incremental changes (>5%) from 80% of the Magstim 200's maximal capacity output, indicative of a 'peak' response.